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Life Systems, Inc., Cleveland, OH

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16. ABSTRACT

This document provides the health effects basis to be considered in establishing the MCLG for radon. To achieve this objective, data on pharmacokinetics, human exposure, acute and chronic toxicity to animals and humans, epidemiology and the mechanisms of toxicity were evaluated. Specific emphasis is placed on literature data providing dose-response information. Thus, while the literature search and evaluation performed in support of this document was comprehensive, only the reports considered most pertinent in the derivation of the MCLG are cited in this document. The comprehensive literature search in support of the analysis of health effects, exposure and occurrence in this document includes information published up to January, 1991; however, more recent information may have been added during the review process.

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FOREWORD

Section 1412 (b) (3) (A) of the Safe Drinking Water Act, as amended in 1986, requires the Administrator of the Environmental Protection Agency to publish maximum contaminant level goals (MCLGs) and promulgate National Primary Drinking Water Regulations for each contaminant, which, in the judgement of the Administrator, may have an adverse effect on public health and which is known or anticipated to occur in public water systems. The MCLG is nonenforceable and is set at a level at which no known or anticipated adverse health effects in humans occur and which allows for an adequate margin of safety. Factors considered in setting the MCLG include health effects data and sources of exposure other than drinking water.

This document provides the health effects basis to be considered in establishing the MCLG for radon. To achieve this objective, data on pharmacokinetics, human exposure, acute and chronic toxicity to animals and humans, epidemiology and mechanisms of toxicity were evaluated. Specific emphasis is placed on literature data providing dose-response information. Thus, while the literature search and evaluation performed in support of this document was comprehensive, only the reports considered most pertinent in the derivation of the MCLG are cited in this document. The comprehensive literature search in support of the analysis of health effects, exposure and occurrence in this document includes information published up to January, 1991; however, more recent information may have been added during the review process.

When adequate health effects data exist, Health Advisory values for less than lifetime exposure (One-day, Ten-day and Longer-term, approximately 10% of an individual's lifetime) are included in this document. These values are not used in setting the MCLG, but serve as informal guidance to municipalities and other organizations when emergency spills or contamination situations occur.

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I. SUMMARY

Radon is a naturally occurring radioactive element which arises from the decay of primordial uranium and thorium in the earth's crust. The occurrence of radon in drinking water supplies is governed by the physical and chemical behavior of uranium, thorium, radium and radon in aquifers and surficial deposits (ATSDR 1989). Radon-222 (Rn-222) is the isotope of primary environmental concern and, in this document, the term radon refers to Rn-222. Population-weighted mean concentrations of radon in water supplies are 200 pCi/L for large systems and 600 pCi/L for small systems (Longtin 1988, USEPA 1988). The presence of radon in drinking water supplies leads to exposure by the oral route (ingesting water containing radon) and also by the inhalation route (breathing air containing radon released from water used in the house).

Radon ingested in water is absorbed from the small intestine into the bloodstream and transported to the lung where it is exhaled (Suomela and Kahlos 1972, Hess and Brown 1991). Approximately 1% of ingested radon decays while in the body (Hursh et al. 1965). Ingestion of short-lived radon decay products in water is not expected to be of significant concern compared to ingestion of radon. Crawford-Brown (1990) has developed a biokinetic model to calculate organ-specific radiation doses following ingestion of radon.

Radon in potable water supplies leads to inhalation exposure to radon gas and radon decay products due to release of radon gas into the air from household uses of water (USEPA 1991b). The dose to sensitive cells of the lung from inhalation of radon decay products is not calculated directly, but toxicokinetic considerations are important for comparing the exposure conditions in mines and homes. The concentration of radon decay products in air has been designated with a special unit, the Working Level (WL), and the unit for cumulative exposure to radon decay products is Working Level Months (WLM). The BEIR IV committee of the National Academy of Sciences considered that the radiation dose to the lung received in a mine and in a home would be nearly equal for a given measured radon decay product concentration in WLM. This near-equality results from an approximate balancing of the effects of the smaller particles and greater unattached fraction in a home that would make the dose higher and of the slower breathing rate that would make the dose lower (NAS 1988). A recent reevaluation of the relative dosimetry of radon decay products in mines and homes has suggested that the dose may be 20-30% lower in homes (NAS 1991); however, current risk estimates are based on the BEIR IV analysis (NAS 1988). The dose to body organs from inhalation of radon gas is estimated using the RADRISK model developed by USEPA (Sullivan et al. 1981).

The occurrence of radon in ground water supplies follows an approximately log-normal distribution, with a mean value of about 650 pCi/L (Longtin 1988, USEPA 1988). These data can be used to estimate population exposure from community water supplies and can be extrapolated to estimate exposure from nontransient non-community water supplies (USEPA 1990a). Approximately 800,000 people are exposed to water containing more than 2,000 pCi/L of radon, 650,000 from community water supplies and 150,000 from nontransient non-community water supplies (USEPA 1990a). Approximately 17,000,000 people are exposed to water containing more than 300 pCi/L of radon in community and nontransient non-community water supplies (USEPA 1991a). Another source of radon exposure by inhalation is the accumulation of radon from soil gas in indoor air. Radon from water supplies contributes a mean of about 5% of the indoor air radon (USEPA 1990a). Radon from water supplies is the only source of ingestion exposure to radon.

Human exposure to radon in water supplies occurs due to ingestion of radon-containing water and inhalation of radon released from water from household uses such as showers, washing machines, etc. Ingestion exposure is evaluated based on consumption of 1 L/day of direct tap water (water consumed directly from the tap without heating or mixing), which loses 20% of its initial radon content during the processes of filling a glass and drinking the water (USEPA 1991a). Inhalation exposure is evaluated in terms of a water-to-air transfer factor. The transfer factor is the ratio of the water concentration of radon to the average increase in the air concentration of radon attributable to release from water. The national average transfer factor is estimated to be about 10,000:1 to 15,000:1, and to range from about 1,000:1 to 100,000:1 (Becker and Lachajczyk 1984, Nazaroff et al. 1987). The transfer factor approach may underestimate exposure to radon gas because it does not account for peaks of concentration from activities such as showering. The extent of the underprediction of radon gas exposure is typically about 50% (USEPA 1991a). The extent of the underprediction of exposure to radon decay products is likely to be less because of the slower buildup of decay products during showers. However, the detailed dosimetry of radon decay products under showering conditions has not been investigated, and this remains as a substantial source of uncertainty in evaluating inhalation exposure (USEPA 1991a). Use of a transfer factor of 10,000:1 provides a reasonable estimate of typical human exposure to radon and radon decay products released from all household uses of water (USEPA 1991a).

No information was located concerning health effects in animals of ingestion of radon. Prolonged inhalation exposure of rats or dogs to radon and radon decay products causes lung cancer. Hamsters appear to be resistant to the carcinogenic effects of radon. No threshold for lung cancer induction by radon exposure is apparent in animal experiments. Quantitative analyses indicate that risk increases with increasing total exposure, with decreasing exposure rate, with increasing unattached fraction and with decreasing equilibrium factor (NAS 1988).

Epidemiologic studies have clearly established that inhalation exposure to radon and radon decay products causes lung cancer in miners. Dose-response relationships are generally linear, with increased risk of lung cancer often observed even in the lowest exposure groups, although the increases are not always significant in the lowest groups (NAS 1988). Increased risk of lung cancer among populations exposed to radon and radon decay products in home air has been observed in several studies (Samet 1989). Studies examining incidences of other types of cancer among populations exposed to radon in drinking water have found mixed results (Hess et al. 1983, Collman et al. 1988, 1991). Children may be at higher risk of developing lung cancer later in life than adults exposed to equal concentrations of radon decay products (ICRP 1987). Smokers are at substantially higher risk than nonsmokers for developing lung cancer from exposure to radon decay products (NAS 1988).

The harmful effects of ionizing radiation are believed to be the result of damage to DNA and other cellular components caused by chemical reaction with ionized molecules, particularly active oxygen species. Inhalation of radon decay products results primarily in radiation exposure to the lung (NAS 1988). The dose of radiation resulting from exposure to radon gas by ingestion or inhalation varies from organ to organ. Models have been developed to calculate the rate at which radiation is absorbed in a given organ, based on the concentration of radioactive elements in that organ and in surrounding organs, the frequency with which each element decays, the energy of each decay event and the fraction of emitted energy that is absorbed in the particular organ (USEPA 1989b, Crawford-Brown 1990).

Quantification of the toxicological effects of radon in drinking water supplies considers separately the risk from ingestion of radon, the risk from inhalation of radon decay products and the risk from inhalation of radon gas.

The risk from ingestion of radon was evaluated assuming lifetime ingestion of 1 L/day of direct tap water from which 20% of the radon is lost during the process of filling a glass and drinking the water. Organ-specific radiation doses following radon ingestion were calculated using the model of Crawford-Brown (1990) and organ-specific risk coefficients were derived from quantitative evaluation of epidemiological data on human cancer risk following exposure to several types of ionizing radiations (USEPA 1989b). Lifetime risks from radon ingestion per pCi/L of radon in water are estimated to be 1.5E-7 for fatal cancers and 2.2E-7 for total cancers.

The risk coefficient for inhalation of radon decay products was derived from quantitative analyses of epidemiologic studies on underground miners by two expert committees, the ICRP 50 committee (ICRP 1987) and the BEIR IV committee (NAS 1988). As a central estimate of risk from indoor radon exposure, USEPA uses a value of 360 deaths per million person-WLM of exposure with an estimated range of 140-720 deaths per million person-WLM (USEPA 1989b). The risk from inhalation of radon decay products was evaluated assuming a transfer factor of 10,000:1, 75% home occupancy and 50% equilibrium between radon gas and radon decay products. The calculated risks from inhalation of radon decay products corresponding to 1 pCi/L of radon in water are estimated to be 4.9E-7 for fatal cancers and 5.1E-7 for total cancers.

The risk from inhalation of radon gas was evaluated using organ-specific doses calculated using the RADRISK model, a metabolic compartmental model, which calculates absorption, distribution, metabolism and excretion of radon and radon decay products (Dunning et al. 1980, Sullivan et al. 1981). Organ-specific risk coefficients were derived from quantitative evaluation of epidemiological data on human cancer risk following exposure to several types of ionizing radiations (USEPA 1989b). Inhalation of radon gas was quantified using the assumptions of a transfer factor of 10,000:1, 75% home occupancy and a breathing rate of 22,000 L/day. The excess lifetime risks from inhalation of radon gas predicted by the RADRISK model per pCi/L of radon in water are 2.0E-8 for fatal cancers and 3.0E-8 for total cancers.

Adding together fatal cancer risks for ingestion of radon (1.5E-7 per pCi/L), inhalation of radon decay products (4.9E-7 per pCi/L) and inhalation of radon gas (2.0E-8 per pCi/L) yields a fatal cancer risk of 6.6E-7 per pCi/L. Similarly, adding total cancer risk for ingestion of radon (2.2E-7 per pCi/L), inhalation of radon decay products (5.1E-7 per pCi/L) and inhalation of radon gas (3.0E-8 per pCi/L) yields a total cancer risk of 7.6E-7 per pCi/L. The concentrations in drinking water corresponding to various levels of excess lifetime risk are displayed as follows:

Excess cancer risk	Drinking water concentration of radon (pCi/L)
Total Cancers	
10^{-4}	130
10^{-5}	13
10^{-6}	1.3
Fatal Cancers	
10^{-4}	150
10^{-5}	15
10^{-6}	1.5

Uncertainties in evaluating the health risks of radon in water supplies were analyzed. One of the largest sources of uncertainty is the assessment of exposure to radon decay products in air resulting from the presence of radon in water supplies. Uncertainty in this assessment includes estimation of the transfer of radon gas from water to air, structural and human activity parameters influencing exposure in general household air and exposure to peaks of concentration in showers, and dosimetric assumptions used to extrapolate observed risks in underground mines to household exposure. There does not appear to be any systematic bias towards over- or under-prediction of risk, due to these or other assumptions and parameters. The uncertainty contributed by modelling assumptions and parameter variability in prediction of average population exposure may be a factor of 3 to 5 and the uncertainty in extrapolation of lung cancer risk may be a factor of 3. These considerations suggest that the true average individual risk is likely to be within an order of magnitude in either direction of the risks calculated for members of the general population.

II. PHYSICAL AND CHEMICAL PROPERTIES

A. Chemical Properties - Elemental

Radon is a noble gas and is therefore chemically inert. Its melting point is -71°C and its boiling point is -61.8°C (ATSDR 1989). Its solubility in water is 230 cm^3/L at 20°C (ATSDR 1989). In a closed system at equilibrium, the radon concentration in air (in pCi/L) will be about 4 times the radon concentration in water (in pCi/L) at 20°C (von Dobeln and Lindell 1964). Radon is produced in soil by the decay of radioactive precursors in minerals, and this process generally transfers radon into air- or water-filled soil pore spaces by alpha recoil (Michel 1990). The radon diffuses or is transported by air or water flow in the soil, and eventually either decays to chemically-reactive decay products or reaches the atmosphere. The amount of radon produced by parent isotopes in soil and rock and the competing fates of decay and transport determine the amount of radon dissolved groundwater in a given aquifer (ATSDR 1989).

B. Physical Properties

Radon has three naturally-occurring isotopes, radon-222 (Rn-222), radon-220 (Rn-220) and radon-219 (Rn-219). Each is the product of a radioactive decay series beginning with a long-lived parent isotope, uranium or thorium. The parent isotope undergoes a spontaneous transformation that results in the emission of a radioactive particle (alpha or beta particle) plus radiant energy (gamma rays) and the formation of a radioisotope of a different element. This process is repeated until a stable isotope of lead is formed.

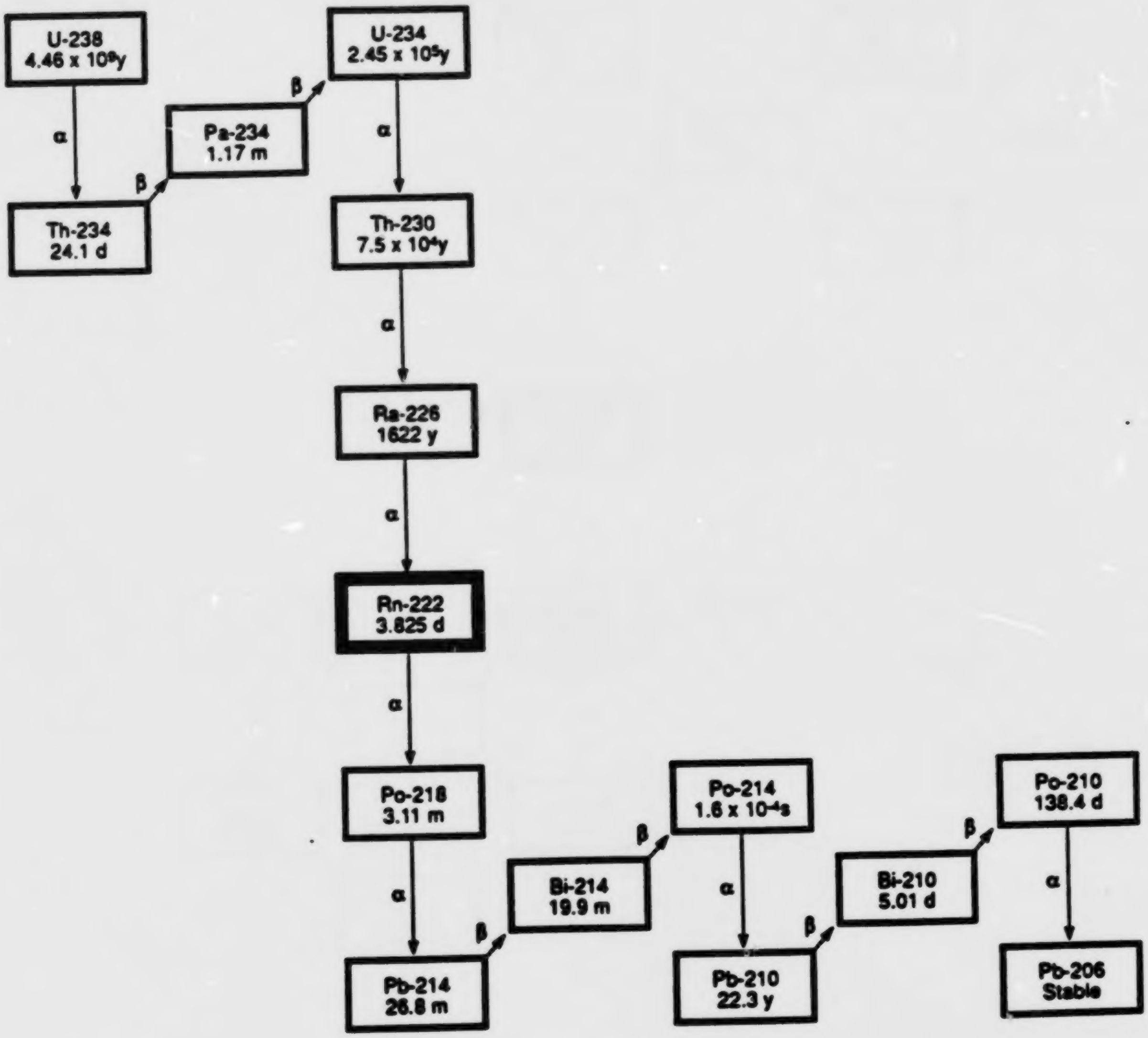
Figures II-1 to II-3 summarize the decay series of uranium and thorium isotopes, showing the sources and decay products of naturally-occurring radon isotopes. The alpha or beta particles emitted during decay are indicated, and also the half lives of each isotope. Rn-222 is the isotope of primary environmental concern, because the much shorter half lives of Rn-220 and Rn-219 mean that their most likely fate is to decay close to their site of formation in soil (ATSDR 1989). In this document, radon is used to refer to Rn-222. The short-lived decay products of radon refer to Po-218, Pb-214, Bi-214 and Po-214, while the long-lived decay products are Pb-210, Bi-210 and Po-210.

C. Occurrence

High levels of radon may occur in ground-water supplies from granitic aquifers, particularly in New England and the Appalachian Highlands/Piedmont region in the United States (Hess et al. 1985). The radon activity in surface waters is generally very low (Hess et al. 1985). The National Inorganics and Radionuclides Survey found radon activity exceeding 100 pCi/L in 72% of groundwater sources surveyed, with highest values occurring in sources serving the smallest populations (Longtin 1988, USEPA 1988). The maximum measured value was 25,700 pCi/L. Population-weighted mean concentrations were 200 pCi/L for sources serving more than 1,000 people and 600 pCi/L for sources serving less than 1,000 people (USEPA 1988).

D. Summary

Radon is a naturally occurring radioactive element which arises from the decay of primordial uranium and thorium in the earth's crust. The occurrence



Uranium-238 Series

Figure II-1 Radioactive Decay Series Showing Precursors and Decay Products of Radon-222

Thorium-232 Series

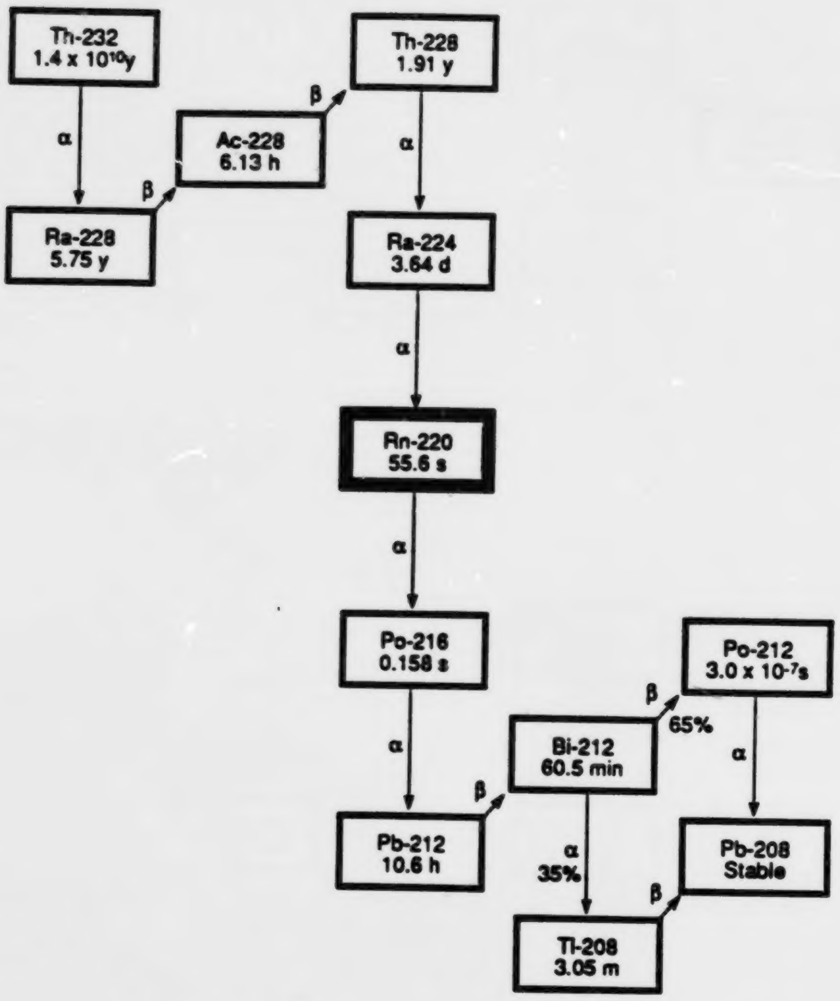


Figure II-2 Radioactive Decay Series Showing Precursors and Decay Products of Radon-220

Uranium-235 Series

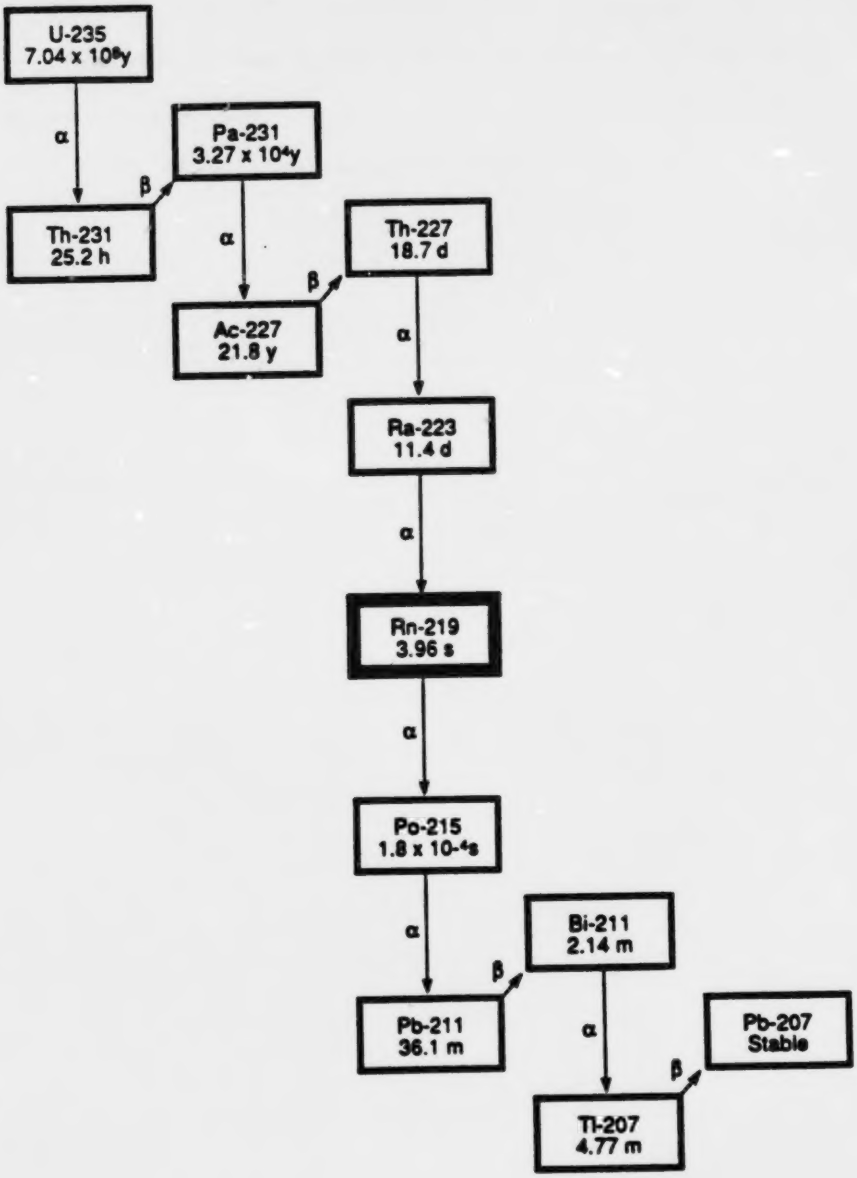


Figure II-3 Radioactive Decay Series Showing Precursors and Decay P Products of Radon-219

of radon in drinking water supplies is governed by the physical and chemical behavior of uranium, thorium, radium and radon in aquifers and surficial deposits (ATSDR 1989). Figures II-1 to II-3 summarize the decay series of uranium and thorium isotopes, showing the sources and decay products of radon isotopes. Rn-222 is the isotope of primary environmental concern. Population-weighted mean concentrations are 200 pCi Rn-222/L for large systems and 600 pCi Rn-222/L for small systems (Longtin 1988, USEPA 1988).

III. TOXICOKINETICS

A. Ingestion Exposure

Investigations of the toxicokinetics of radon ingested by humans have generally involved measuring exhaled radon and/or whole-body gamma emission following ingestion of radon by volunteers (Hursh et al. 1965, Suomela and Kahlos 1972, von Dobeln and Lindell 1964, Gosink et al. 1990, Hess and Brown 1991). These studies have established that radon ingested in water is absorbed into the bloodstream from the small intestine and circulates to the lungs, from which it is exhaled (Hursh et al. 1965). The biological half-life of radon in the body ranges from 30 to 50 minutes (Suomela and Kahlos 1972, Hess and Brown 1991). No study using radon has included measurements of more than a few organ contents, but Correia et al. (1987) used xenon as a surrogate for radon in a study sponsored by the U.S. Environmental Protection Agency (Crawford-Brown 1989). A gamma camera was used to follow the movement of xenon through the bodies of 22 male and female adult volunteers for up to 10 hours after drinking water containing millicurie levels of xenon. During the initial postingestion period, images were taken at 1 second intervals to determine initial rapid escape of xenon. The concentration of xenon in various organs was calculated from the measured areal density using organ masses and volumes of the "Reference Man" (ICRP 1975), based on the areal density in the stomach immediately after ingestion, when the xenon content was assumed to be equal to the entire amount ingested (Crawford-Brown 1989). Xenon concentration was converted to radon concentration using the ratio of tissue/blood partition coefficients (Crawford-Brown 1989).

Crawford-Brown (1989, 1990) analyzed the data from the Correia et al. (1987) study on average organ radon retentions as a function of time after ingestion in two ways. The first analysis fitted exponential or multiexponential retention functions for eight organs (stomach, small intestine, ascending colon, descending colon, liver, muscle, fat and whole body). These empirical retention functions are listed in Table III-1. The second analysis was based on measured organ washout half-times following inhalation of xenon, shown in Table III-2 and on a general biokinetics model developed for ingested radon. This model is shown schematically in Figure III-1, and compartmental masses and volumes used in the model are listed in Table III-3 (ICRP 1975). Crawford-Brown (1990) derived rate constants for this model that gave the best fit to the measured concentrations and washout half-times for the eight organs. The fitted rate constants are listed in Table III-4. Crawford-Brown (1990) found that calculated organ radiation doses differed less than 10% between those calculated using the empirical retention functions and those calculated using the biokinetics model.

Particular issues addressed by Crawford-Brown (1990) were the behavior of radon in the intestinal walls and lung tissue, and the biokinetics of long-lived decay products. In the intestinal walls, a concentration gradient will be established across the lumen between the radon concentration in the gastrointestinal tract and the radon concentration in the bloodstream of the epithelial layer. The concentration of radon is always at least a factor of 10 lower in the bloodstream than in the intestine, and due to the high diffusion coefficient of radon, the concentration profile can be assumed to be linear across the lumen. Crawford-Brown (1990) assumed that the intestinal

Table III-1 Empirical Organ Retention Functions

Organ	Concentration Following Ingestion of 1 pCi Radon (pCi/cm^3)
Stomach	$C(t) = 0.002[0.998 \exp(-0.029t) + 0.002 \exp(-0.0026t)]$
Small Intestine	$C(t) = 0.0002[0.9 \exp(-0.02t) + 0.1 \exp(-0.0045t)] \{1 - \exp(0.099t)\}$
Ascending Colon	$C(t) = 0.0001[0.85 \exp(-0.015t) + 0.15 \exp(-0.006t)] \{1 - \exp(0.099t)\}$
Descending Colon	$C(t) = 0.00003[\exp(-0.0048t)] \{1 - \exp(-0.099t)\}$
Liver	$C(t) = 0.00003[0.87 \exp(-0.022t) +0.13 \exp(-0.0039t)]$
Muscle	$C(t) = 0.00001[\exp(-0.0029t)] \{1 - \exp(0.23t)\}$
Fat	$C(t) = 0.00001[\exp(-0.005t)] \{1 - \exp(0.099t)\}$
Whole Body	$C(t) = 0.0001\{0.92 \exp(-0.022t) + 0.08 \exp(-0.0035t)\}$
General Tissue	$C(t) = 0.000005[\exp(-0.005t)] \{1 - \exp(-0.099t)\}$

Adapted from Crawford-Brown 1990

Table III-2 Measured Xenon Organ Washout Half-times

Organ	Half-time (Minutes)
Liver, Spleen and Kidneys	1.3
Stomach and Small Intestine	1.6
Upper and Lower Large Intestine	4.3
Fat Tissue	362

Adapted from Crawford-Brown 1990

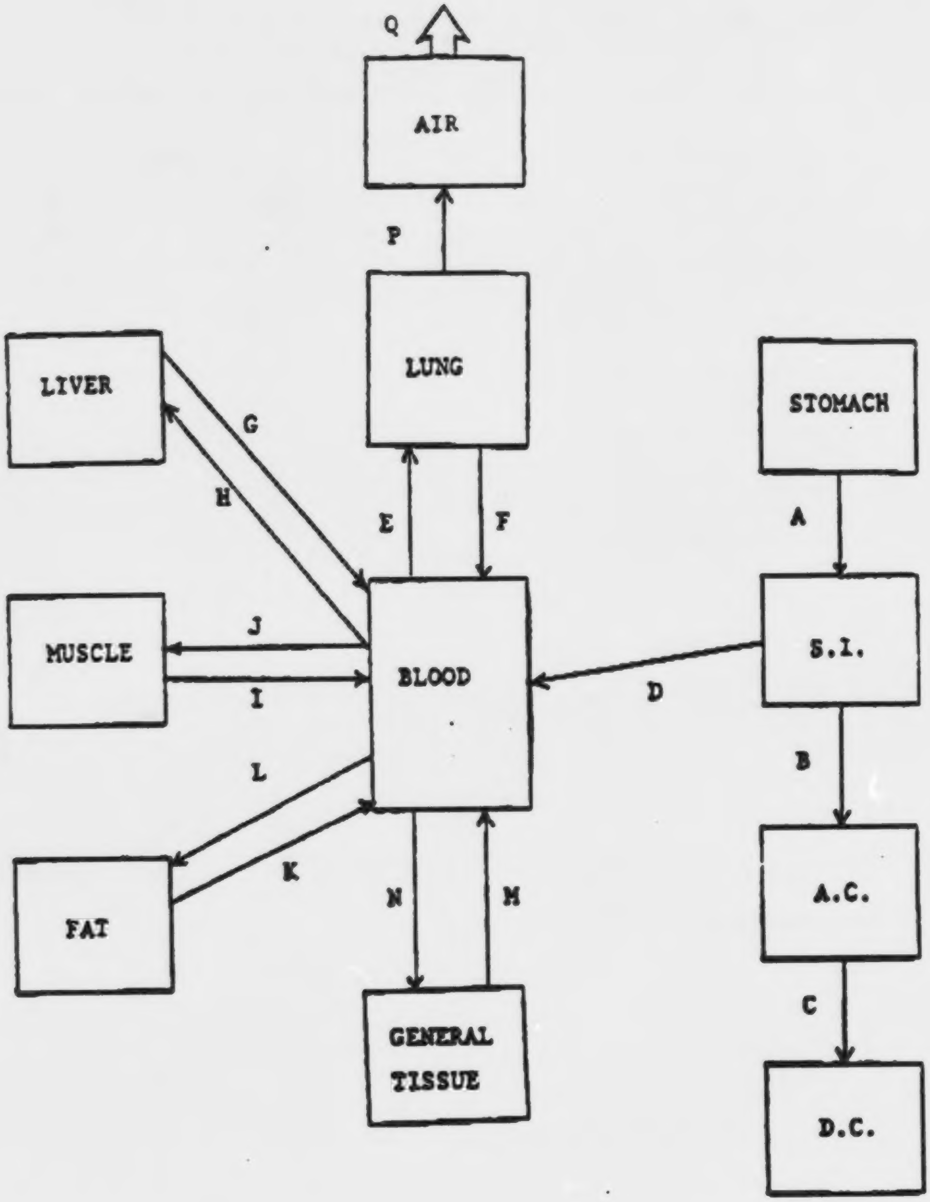


Figure III-1 General Biokinetics Model for Ingested Radon

Table III-3 Compartmental Parameters in the Biokinetic Model for Radon Ingestion

Compartment	Value
Stomach with Contents	400 g
Small Intestine	1,040 g
Upper Large Intestine	430 g
Lower Large Intestine	295 g
General Tissue	32,035 g
Blood (plasma)	3,100 g
Blood (total)	5,500 g
Liver	1,800 g
Lung Airspace	4,300 cm^3
Lung Minute Volume	7,500 cm^3/min
Fat	13,500 g
Muscle	28,000 g

Adapted from Crawford-Brown 1990

Table III-4 Fitted Transfer Rate Constants for the General Biokinetics Model

Pathway in Fig. 1	From	To	Rate Constant (min^{-1})
A	Stomach	Small Intestine	0.04
B	Small Intestine	Ascending Colon	0.005
C	Ascending Colon	Descending Colon	0.005
D	Small Intestine	Blood	1.5
E	Blood	Lung	0.16
F	Lung	Blood	0.4
G	Liver	Blood	0.4
H	Blood	Liver	0.13
I	Muscle	Blood	0.0073
J	Blood	Muscle	0.057
K	Fat	Blood	0.0019
L	Blood	Fat	0.083
M	General Tissue	Blood	0.4
N	Blood	General Tissue	1.8
P	Lung	Alveolar Air	0.49
Q	Alveolar Air	Exhaled Air	1.74

Adapted from Crawford-Brown 1990

lumen is 40 μm thick and that the radiosensitive crypt cells are 30 μm below the surface in calculating radiation doses to the stomach, small intestine, ascending colon and descending colon.

The data of Correia et al. (1987) did not permit Crawford-Brown (1990) to separate radon in lung tissue from radon in air of the alveolar region. Lung tissue radon content was assumed to be equal to liver radon content. Lung tissue is irradiated not only by radon decaying within the tissue, but also by radon decaying in the air of the alveolar sacs during exhalation. Crawford-Brown (1990) assumed that Po-218 rapidly deposits on alveolar walls and that subsequent decay products either decay or are removed by mucociliary clearance. The calculated radiation dose from alveolar decay was about 25% of the dose from decay in the lung tissue.

No direct measurements of the biokinetics of radon decay products in the body were available. Crawford-Brown (1990) assumed that short-lived decay products remained at the site of their production, and showed that consideration of their recirculation would affect calculated doses by less than 10%. For Pb-210, with a half-life of 22.3 years, Crawford-Brown (1990) calculated the bone marrow and surface doses assuming that 1% of ingested radon decays to Pb-210 in the body and that 55% of this lead deposits in bone. The doses to bone surface and marrow from Pb-210 decay were about 7 and 100 times less than to general body tissue from radon decay. This implies that Pb-210 does not make a substantial contribution to the dose from radon ingestion. Crawford-Brown (1990) also considered pre-existing decay products ingested in water. The short-lived decay products from Po-218 to Po-214 were shown to pose much less of a risk at equal activities (Crawford-Brown 1990). The long-lived decay products from Pb-210 to Po-210 may pose risk, but are likely to be geologically separated from radon due to their differing chemical properties (Crawford-Brown 1990).

Crawford-Brown (1990) considered the reduction in dose to the stomach wall if all short-lived decay products were swept away into the blood stream prior to their radioactive decay. This reduction would be by a factor of 3, because two of the alpha decays are from short-lived decay products. However, for decay products within the intestinal lumen, the physiologic process of diffusion rather than circulatory transport is more likely to remove decay products, which may be sufficiently slow that much of the Po-218 (half-life 3.05 minutes) will undergo radioactive decay within the lumen (Hursh et al. 1965). Physiologic removal of decay products beyond Po-218 would reduce the dose to 2/3 that calculated assuming total retention of decay products.

B. Inhalation Exposure

The inhalation toxicokinetics of radon and radon decay products are considered separately. Lung cancer risk estimates for radon decay products are available from direct human epidemiologic data on underground miners (see Section VIII.B.2), and consideration of the toxicokinetics of radon decay products is needed primarily to determine whether doses are comparable in mines and in homes. No such direct evidence is available for radon gas, and so the output of the toxicokinetic evaluation is a dose per unit inhalation in several organ systems, similar to the output for radon ingestion.

1. Inhalation of Radon Decay Products

Analyses of the toxicokinetics of inhaled radon decay products have mostly concerned the radiation dose received by the sensitive cells of the lung, because other tissues receive considerably lower doses (NAS 1988). Risk estimates for inhalation exposure to radon decay products are derived from observed increases in lung cancer among miners exposed to measured concentrations of decay products (see Section VIII.B.2). Toxicokinetic and dosimetric models are useful in evaluating the quantitative extrapolation of risks observed in adult male miners to risks expected in members of the general population, including children.

Quantification of the Air Concentration of Radon Decay Products

Because of the importance of the short-lived decay products to the radiation dose received by the lung, a special exposure unit, termed Working Level (WL), has been developed to describe the concentration of radon decay products in air. The definition of 1 WL is any combination of Po-218, Pb-214, Bi-214 and Po-214 in 1 L of air that results in the ultimate emission of 1.3×10^5 MeV of alpha-particle energy (NAS 1988). This unit was developed for occupational safety, and for historical reasons exposure duration was quantified in working hours per month. Thus, an additional unit has been defined, the Working Level Month (WLM), which is exposure to 1 WL for 170 hours.

When pure radon is initially released into air, no decay products will be present, the WL is zero and the equilibrium factor is 0. In a closed system, the concentration of decay products will build up, and after about 2 hours, radon will be close to radiologic equilibrium with its short-lived decay products (i.e., the equilibrium factor is close to 1.0) (Becker and Lachajczyk 1984). At equilibrium between radon and its short-lived decay products, 100 pCi/L of radon gas in air corresponds to 1 WL (NAS 1988). Ventilation of a room prevents equilibrium from being achieved, and the actual equilibrium factor depends upon ventilation rate, aerosol size and concentration, and other factors (USEPA 1991d). Measured equilibrium factors range from 0.02 to 0.83 (Toohey et al. 1987). Based on the results of surveys conducted in the United States and other countries, the mean value of the equilibrium factor appears to lie in the range of 0.3 to 0.6 (USEPA 1991d). The use of a nominal point estimate of 0.5 appears to be supported by current data, with an uncertainty range of 0.35-0.55 (USEPA 1991d).

Using a value of 0.5 for the equilibrium factor, the conversion factor is 200 pCi/L of radon gas in air to 1 WL, or 1 pCi/L of radon in air to 0.005 WL (USEPA 1989b). To quantify cumulative exposure to radon decay products, the number of hours per day of exposure must be defined. Home occupancy 18 hours per day (75% of the time) is 540 hours per month, about 3 times the "occupational" month of 170 hours used to define the WLM. Home exposure to 1 WL at 75% occupancy results in exposure to 3.2 WLM/month and 39 WLM/year. Home exposure to 1 pCi/L of radon in air at 75% occupancy results in exposure to 0.19 WLM/year.

Dosimetry of Radon Decay Products

When an atom of radon decays in air, the resulting product atoms exist as charged, reactive ions. These ions are rapidly surrounded by molecules of water or other solvent vapors and become ultrafine aerosols (NAS 1991). The ions in the ultrafine aerosols may remain unattached, may react with aerosol particles or may plate out on walls or other room surfaces. Successive decay products may become detached from aerosols or wall surfaces by alpha recoil. The proportion of decay products in air that exist as ions in ultrafine aerosols rather than attached to larger aerosol particles is important to the dosimetry of radon decay products, and this proportion is called the unattached fraction (NAS 1988). The unattached fraction depends primarily upon the number and size of aerosol particles in the air, and measured values of the unattached fraction range from 0.004 to 0.15 (Reineking et al. 1985). Models suggest that the radiation dose to the lung increases linearly with unattached fraction (NAS 1988). A measured WL counts both unattached decay products and decay products attached to aerosol particles, so the unattached fraction is independent of the WL (NAS 1988). However, room ventilation both increases the unattached fraction and decreases the equilibrium factor (thereby decreasing the WL corresponding to a measured radon concentration in pCi/L). Since an increased unattached fraction and a decreased WL have opposite effects on lung radiation dose, model calculations suggest that the dose corresponding to a measured radon concentration in pCi/L is relatively constant over a range of actual room conditions (USEPA 1991d).

Detailed radon decay product dosimetry models based on a Weibel compartmental model for the lung have been developed by three main groups, Harley-Pasternak (H-P), Jacobi-Eisfeld (J-E) and James-Birchall (J-B) (NAS 1988). Each model calculates dose to the tracheobronchial region of the lung, where most lung cancers in humans originate. The BEIR IV committee considered published results of these models to analyze the ratio of the dose per WLM in mines and homes (NAS 1988). The factors that were considered were particle size, unattached fraction, equilibrium factor and breathing rate. Lung morphology, particle deposition rate, mucus thickness and depth of target cells were assumed to be comparable in miners and the general population.

For respirable particles, all three dosimetry models indicate that the mean dose to the tracheobronchial region increases with decreasing particle size, due to the greater penetration and retention of small particles. The BEIR IV committee assumed that the activity mean diameter was 0.12 μm in homes and 0.17 μm in mines, and concluded that the smaller particle size in homes would result in a 1.4 times higher dose at a constant WLM in homes than in mines (NAS 1988).

For unattached fraction, all three dosimetry models show a linear increase in dose with increasing unattached fraction, but the slopes of the lines differ. The BEIR IV committee assumed that the unattached fraction was 0.04 in mines and 0.07 in homes, and concluded that the higher unattached fraction in homes would result in a dose that was 1.0 to 1.4 times higher (mean 1.2 times higher) dose at a constant WLM in homes than in mines (NAS 1988).

For the equilibrium factor, the BEIR IV committee cited the results from Jacobi and Eisfeld (1982) indicating that with constant total alpha energy (i.e., constant WL), the equilibrium factor has little effect on tracheobronchial dose. This is primarily due to the similar behavior of the short-lived decay products, because if WL is constant, then the equilibrium factor only influences the relative proportion of the successive decay products, not the total potential alpha energy. On this basis, the BEIR IV committee concluded that variations in the equilibrium factor would not influence the relative dose received in the mine and in the home for exposure expressed in WLM (NAS 1988).

Breathing rates would be expected to differ substantially between miners engaged in moderate to heavy activity and the general population engaged in mostly light activity or sleeping in a home. All three dosimetry models indicate that increased breathing rate increases the dose per WLM, although less than linearly because faster breathing allows less time for particles to deposit in the airways (NAS 1988). The BEIR IV committee assumed that the breathing rate was 30 L/min for miners and 12.5 L/min for average people in a home, and used the dosimetric models to estimate that the dose in a home was 0.56 times as high as the dose in a mine at a constant WLM (NAS 1988). One limitation cited in that analysis was the lack of information on the ratio of nose to mouth breathing in mines or in homes (NAS 1988). About half of the unattached fraction deposits in the nose during nose breathing, while little or none deposits in the mouth during mouth breathing. If miners do a significantly higher amount of mouth breathing than average people in homes, the differences between the doses would be even greater than 0.56 (NAS 1988).

The BEIR IV committee concluded that the increase in dose per unit exposure in homes from smaller particles and higher unattached fraction would be essentially cancelled by the decrease in dose from decreased breathing rate, and that at the present state of knowledge, the dose to target cells from a given radon decay product concentration in WLM should be considered equivalent in mines and in homes (NAS 1988). An expanded analysis of this issue was recently completed (NAS 1991). This analysis made a more detailed assessment of the effects of the four parameters previously analyzed (sources and characteristics of aerosols in mines and homes, parameters determining the unattached fraction and equilibrium factor and breathing rate and route as a function of physical activity level). This analysis also considered the consequences of various underlying model assumptions, including the location of cells at risk, changes in lung physiology from occupational exposures and/or smoking, the efficiency of nasal and bronchial deposition, the solubility of decay products in mucus and the hygroscopic growth of aerosols in the respiratory tract. This latter factor had the greatest effect on model results, and the assumption that aerosol particles double in size in the respiratory tract due to accretion of water significantly reduces the calculated dose to target cells in homes compared to mines (NAS 1991). USEPA is in the process of analyzing this report and will consider these factors in determining whether to adjust the risk coefficients for prediction of risk from inhalation of radon decay products in household air.

2. Inhalation of Radon Gas

The toxicokinetic model for inhaled radon gas uptake and retention that is the biokinetic portion of the RADRISK model used by USEPA is derived from the Oak Ridge National Laboratory (ORNL) model for noble gas metabolism (Bernard and Snyder 1975). Retention is calculated based on a five-compartment model approximated as a matrix with cross terms representing inter-compartmental transfer. Activity concentrations in tissues are based on studies performed by Nussbaum (1957). Dosimetric estimates are based on tissue concentrations and tissue energy absorption (Dunning et al. 1980).

Toxicokinetics of radon decay products that are produced in the body from inhaled radon gas are evaluated in the RADRISK model using element-specific parameters. The RADRISK model differs from ICRP models in general in the treatment of radioactive decay products produced in organs and tissues (Dunning et al. 1984). ICRP models assume that all radioactive decay products generated in the body follow the metabolic behavior of the precursor radionuclide (ICRP 1979). In contrast, the RADRISK model assumes that the retention of a decay product in an organ or tissue is governed by the metabolic properties of that element. Therefore, to calculate the ionizing energy deposition in tissues associated with inhalation of radon gas, not only is the metabolic model for radon used, but retention parameters for all radon decay products must be evaluated and energy deposition summed within tissues to calculate the total absorbed radiation dose in the various tissues. Calculating the radiation dose from deposited and retained radon requires use of toxicokinetic models for all the elements in the radioactive decay chain of Rn-222 (see Figure II-1).

The general biokinetic model for radon decay products is derived from the ICRP20 model for alkaline earth metabolism (ICRP 1973). The ICRP task group developed the model using human and animal data on alkaline earth intake, retention and excretion (ICRP 1973). Adams et al. (1978) made a functional fit to the tabulated retention values in ICRP20, while holding constant the area under the retention curve. Since these retention functions avoided the necessity of evaluating the incomplete gamma function in the ICRP20 model, they have been used in USEPA biokinetic modeling (Sullivan et al. 1981, Dunning et al. 1984).

Parameters needed for the general biokinetic model are absorption of the element from the intestine (f_1), fractional uptake into various tissues (f_2') and retention of the element in tissues. Radon decay products enter the gastrointestinal tract and the blood from the lung. The gastrointestinal tract portion of the model used to evaluate exposure to radon has four compartments: stomach, small intestine, upper large intestine and lower large intestine. Rates of transfer, expressed as organ volumes transferred per day, are: stomach to small intestine, 24/day; small intestine to upper large intestine, 6/day; upper large intestine to lower large intestine, 1.85/day; and removal from lower large intestine, 1/day (Sullivan et al. 1981). Elements are absorbed into the blood only from the small intestine, according to values of f_1. After absorption into the blood, radon decay products are distributed among tissues, according to values of f_2'.

The calculation of organ and tissue retention of radon decay products accounts both for biological removal by exchange and excretion and for physical removal by radioactive decay. In general, retention is defined as the amount of an element remaining in a tissue or organ at some time after the intake of the element. Removal of the element by biological processes is assumed to be first order, so that the net amount removed at any given time is directly proportional to the amount in the tissue at that time. This assumption means that biological removal can be characterized by a biological half life, T_B, which is the time taken to remove half of the concentration initially present. Experimental studies have shown that many elements have a number of different half lives in a given tissue or organ, each corresponding to a different fraction of the element initially present. For example, lead is estimated to have three compartments in bone, with biological half lives ranging from 12 to 12,000 days (Adams et al. 1978). The numerical values for the number of compartments and their biological half lives are usually determined in studies where there is a known intake of a radionuclide. The whole body or specific organs or tissues are radioassayed, to determine the amount of activity present at various times after intake. This data is used to develop a retention curve from which the numerical values of parameters can be obtained. Metabolic studies to determine retention parameters are usually done in experimental animals. However, for some radionuclides there is metabolic data from both humans and animals.

For a given element i, retention in organ s at time t is given by:

$$R_{is}^{E}(t) = \sum_{j=1}^{L_{is}} F_{ijs}\exp(-(\lambda_i^{R} + \lambda_{ijs}^{B})t)$$

where:

R_{is}^{E} = the effective retention of element i in organ s at time t (dimensionless).

L_{is} = the number of biological compartments for element i in organ s, up to a maximum of 5 (dimensionless).

F_{ijs} = the fraction of element i residing in the jth compartment in organ s (dimensionless).

λ_i^{R} = the rate of removal of element i by radioactive decay in $(\text{days})^{-1}$, calculated as $(\ln 2)/T_i^{R}$, where T_i^{R} is the radioactive half life of element i in days.

λ_{ijs}^{B} = the rate of biological removal of element i from compartment j of organ s in $(\text{days})^{-1}$, calculated as $(\ln 2)/T_{ijs}^{B}$, where T_{ijs}^{B} is the biological half life of element i in compartment j of organ s in days.

t = time in days.

Table III-5 lists the biokinetic parameters for the elements in the radon (Rn-222) decay series: polonium, bismuth and lead (see Figure II-1). The RADRISK model actually also includes the small contributions from minor branching products (USEPA 1989b), such as astatine-218, representing 0.019% of Po-218 decays, and thallium-210, representing 0.021% of Bi-214 decays (NAS 1988).

The concentration of radon and radon decay products in the body is calculated for a constant inhalation of 1 pCi/yr of radon gas (Dunning et al. 1980, Sullivan et al. 1981). The rate of change of the concentration of radon decay products in a given compartment of a given organ is calculated as the difference between its inflow (from production by radioactive decay of its immediate precursor plus transfer from the respiratory tract, gastrointestinal tract and blood) and its loss (through biological removal and radioactive decay). Values for the rate of change of concentration are integrated to yield the concentration of each element in each organ system as a function of time in 1 year intervals (Sullivan et al. 1981). These values are passed to another module of the RADRISK program, which calculates organ-specific doses of high-LET and low-LET radiation corresponding to the calculated organ-specific concentration of radionuclides (see Section VII.B.).

The RADRISK model assigns the metabolic behavior of radon decay products on the basis of elemental form, in contrast to the ICRP model which treats all decay products the same as the precursor radionuclide. This difference can have a substantial impact on dose estimates (Dunning et al. 1984). The assumption that retention of decay products is different from retention of the precursor radionuclide is clearly appropriate for soft tissues where elemental

Table III-5 Biokinetic Parameters for Radioisotopes in the Radon Decay Series

Element	Absorption from the Intestine	Organ	Fractional Tissue Uptake	Number of Compartments	Compartment Fraction	Biological half-life, days	References
Polonium	0.1	Liver	0.1	1	1.0	50	ICRP 1979
		Kidney	0.1	1	1.0	50	
		Spleen	0.1	1	1.0	50	
		Other tissues	0.7	1	1.0	50	
Bismuth	0.05	Kidney	0.4	2	0.60 0.40	0.60 5	Adams et al. 1978
		Other tissues	0.6	2	0.60 0.40	0.60 5	
Lead	0.2	Bone	0.55	3	0.60 0.15 0.25	12 180 12,000	Adams et al. 1978
		Liver	0.25	3	0.80 0.18 0.02	12 180 12,000	
		Kidney	0.02	3	0.80 0.18 0.02	12 180 12,000	
		Other tissues	0.18	3	0.80 0.18 0.02	12 180 12,000	
Thallium	0.95	Kidney	0.05	1	0.05	7	Adams et al. 1978
		Other tissues		1	0.95	7	

Adapted from Sullivan et al. 1981

form will govern metabolic behavior in the aqueous phase in tissues. In skeletal tissues, however, the decay products may be produced and trapped in the crystal lattice of the hydroxyapatite. They would, in this situation, continue to have the retention characteristics more like the precursor radionuclide (Neuman and Neuman 1958, McLean and Budy 1964). That portion of the decay products created in skeletal tissues outside the crystal lattice would be expected to behave as their elemental characteristics direct them. Uncertainties influencing the calculations of the organ distribution of inhaled radon gas and radon decay products are discussed in Chapter IX.

C. Summary

Radon ingested in water is absorbed from the small intestine into the bloodstream and transported to the lung where it is exhaled (Suomela and Kahlos 1972, Hess and Brown 1991). Approximately 1% of ingested radon decays while in the body (Hursh et al. 1965). Ingestion of short-lived radon decay products in water is not expected to be of significant concern compared to ingestion of radon. Crawford-Brown (1990) has developed a biokinetic model to calculate organ-specific radiation doses following ingestion of radon.

Radon in potable water supplies also leads to inhalation exposure due to release into the air from household uses of water (USEPA 1991b). The dose to sensitive cells of the lung from inhalation of radon decay products is not calculated directly, but toxicokinetic considerations are important for comparing the exposure conditions in mines and homes. The concentration of radon decay products in air has been designated with a special unit, the Working Level (WL). The BEIR IV committee of the National Academy of Sciences considered that the radiation dose to the lung received in a mine and in a home would be nearly equal for a given measured radon decay product concentration in WL. This near-equality is because the smaller particles and greater unattached fraction in a home that would make the dose higher would be roughly balanced by the slower breathing rate that would make the dose lower (NAS 1988). The relative dosimetry of radon decay products in mines and homes has recently been reevaluated (NAS 1991). The dose to body organs from inhalation of radon gas is estimated using the RADRISK model developed by USEPA (Sullivan et al. 1981).

The influences of uncertainties in toxicokinetics on the quantification of toxicological effects of ingested radon, inhaled radon decay products and inhaled radon gas are discussed in Chapter IX.

IV. HUMAN EXPOSURE

A. Naturally Occurring Levels

1. Radon in Water Supplies

Several surveys of radon occurrence in water supplies have been performed. USEPA (1990a) summarizes recent state-wide surveys in Connecticut, Georgia, New Jersey and Maryland, a system-wide survey in 15 states by the American Water Works Service Co., and the National Inorganics and Radionuclides Survey (NIRS) performed by the Office of Drinking Water (USEPA 1988). The NIRS results were considered the most suitable for deriving estimates of national occurrence because the NIRS sampling program was designed to reflect the national distribution of community ground water supplies (Longtin 1988, USEPA 1988). This survey found a mean radon concentration of about 650 pCi/L, with concentrations exceeding 1,000 pCi/L in 11% of supplies, all of which served fewer than 3,000 people. No similarly-designed surveys were available to estimate radon occurrence in non-community water supplies (e.g., schools) or private wells (USEPA 1990a).

Occurrence distributions were derived from the NIRS data for four size categories of population served: very small (25 - 500), small (501 - 3,300), medium (3,301 - 10,000) and large/very large (>10,000). A log-normal distribution was found to describe the occurrence data, and mean and standard deviation estimates were derived, taking into account the samples reported as below detection limits (100 pCi/L) (USEPA 1990a). The resulting occurrence estimates are presented in Table IV-1 for community ground water supplies. On the assumption that these distributions by size category were similar for

Table IV-1 Estimated Cumulative Occurrence of Radon in Community Ground Water Supplies Based on a Censored Lognormal Distribution Model

System Size (population served)	Number of Systems in the U.S.	Number of supplies with concentrations (pCi/L) of: ≥100	>200	>500	>1,000	>2,000
Very Small						
25-100	17,079	12,327	9,416	5,403	2,978	1,389
101-500	15,354	11,082	8,465	4,857	2,677	1,248
Small						
501-1,000	5,038	2,857	1,792	704	265	78
1,001-3,300	5,185	2,940	1,845	724	273	80
Medium						
3,301-10,000	2,308	1,436	768	181	36	4
Large/Very Large						
10,001-25,000	823	514	273	63	12	1
25,001-50,000	278	174	92	21	4	0
50,001-75,000	77	48	26	6	1	0
75,001-100,000	17	11	6	1	0	0
100,001-500,000	39	24	13	3	1	0
500,001-1,000,000	4	2	1	0	0	0
>1,000,000	0	0	0	0	0	0
Total	46,202	31,415	22,697	11,963	6,247	2,800
Lower Bound		29,834	21,023	10,515	5,118	2,009
Upper Bound		32,996	24,371	13,411	7,377	3,591

Note: Lower and upper bounds are based upon 95% confidence intervals. Subheadings indicate the four system size strata from the NIRS.

Adapted from USEPA (1990a)

community ground water supplies and non-community water supplies, the values in Table IV-2 were derived for non-community water supplies serving nontransient populations. Approximately 6% of community water supplies and 8% of nontransient non-community water supplies have more than 2,000 pCi/L (USEPA 1990a). Approximately 25,900 community and nontransient non-community water supplies have radon concentrations greater than 300 pCi/L (USEPA 1991a).

Approximately 76,000,000 people in the United States are served by groundwater supplies. The populations exposed to various levels of radon were estimated by combining the occurrence estimates in Tables IV-1 and IV-2 with the number of people served (USEPA 1990a). These estimates are given in Tables IV-3 and IV-4. Approximately 800,000 people are exposed to more than 2,000 pCi/L, 650,000 in community water supplies and 150,000 in nontransient non-community water supplies (USEPA 1990a). Approximately 17,000,000 people are exposed to more than 300 pCi/L in community and nontransient non-community water supplies (USEPA 1991a).

2. Radon in Other Media

Because radon is a gas, it would not be expected to be present in food, and no information was located to indicate that radon has ever been detected in food (USEPA 1990a). Radon is present in air due to outgassing from soil and rock. Outdoor air has an average radon concentration of about 0.15 pCi/L, but much higher concentrations may be found in indoor air in homes where soil and ventilation characteristics lead to a large proportion of indoor air being derived from soil air. Several surveys have been completed or are in progress to determine the distribution of indoor air radon concentrations in the United States. Nero et al. (1986) estimated that indoor radon concentrations were

Table IV-2 Estimated Cumulative Occurrence of Radon in Nontransient Non-Community Ground Water Supplies Based on a Censored Lognormal Distribution Model

System Size (population served)	Number of Systems in the U.S.	Number of supplies with concentrations (pCi/L) of:				
		≥100	>200	>500	>1,000	>2,000
Very Small						
25-100	13,899	10,032	7,663	4,397	2,423	1,130
101-500	3,964	2,861	2,185	1,254	691	322
Small						
501-1,000	1,151	653	409	161	61	18
1,001-3,300	205	116	73	29	11	3
Medium						
3,301-10,000	49	30	16	4	1	0
Large/Very Large						
10,001-25,000	4	2	1	0	0	0
25,001-50,000	11	7	4	1	0	0
50,001-75,000	6	4	2	0	0	0
75,001-100,000	2	1	1	0	0	0
100,001-500,000	7	4	2	1	0	0
500,001-1,000,000	0	0	0	0	0	0
>1,000,000	0	0	0	0	0	0
Total	19,298	13,710	10,356	5,847	3,187	1,473
Lower Bound		13,031	9,585	5,129	2,603	1,053
Upper Bound		14,407	11,267	6,565	3,771	1,893

Note: Lower and upper bounds are based upon 95% confidence intervals. Subheadings indicate the four system size strata from the NIRS.

Adapted from USEPA (1990a)

Table IV-3 Estimated Cumulative Population (in Thousands) Exposed to Radon in Drinking Water Exceeding the Indicated Concentrations From Community Ground Water Supplies

System Size (population served)	Number of People (in thousands)	Number of people (thousands) using supplies with concentrations (pCi/L) of:				
		≥100	>200	>500	>1,000	>2,000
Very Small						
25-100	950	686	524	301	166	77
101-500	3,850	2,779	2,123	1,218	671	313
Small						
501-1,000	3,910	2,217	1,391	546	206	60
1,001-3,300	10,000	5,670	3,558	1,397	526	154
Medium						
3,301-10,000	13,310	8,279	4,430	1,047	206	25
Large/Very Large						
10,001-25,000	13,110	8,183	4,355	1,009	194	23
25,001-50,000	9,540	5,955	3,169	734	141	0
50,001-75,000	4,770	2,977	1,584	367	70	0
75,001-100,000	1,360	849	452	105	0	0
100,001-500,000	7,600	4,744	2,524	585	112	0
500,001-1,000,000	2,760	1,723	917	0	0	0
>1,000,000	0	0	0	0	0	0
Total	71,160	44,061	25,025	7,307	2,291	652
Lower Bound		34,718	15,941	2,138	0	0
Upper Bound		53,403	34,110	12,477	4,666	1,503

Note: Lower and upper bounds are based upon 95% confidence intervals. Numbers may not add to totals due to rounding. Subheadings indicate the NIRS system size strata.

Adapted from USEPA (1990a)

Table IV-4 Estimated Cumulative Population (in Thousands) Exposed to Radon in Drinking Water Exceeding the Indicated Concentrations From Nontransient Non-Community Ground Water Supplies

System Size (population served)	Number of People (in thousands)	Number of people (thousands) using supplies concentrations (pCi/L) of: ≥100	>200	>500	>1,000	>2,000
Very Small						
25-100	615	444	339	195	107	50
101-500	937	676	517	296	163	76
Small						
501-1,000	1,015	576	361	141	53	16
1,001-3,300	351	199	125	49	19	5
Medium						
3,301-10,000	273	170	91	22	4	0
Large/Very Large						
10,001-25,000	71	44	24	0	0	0
25,001-50,000	275	172	91	21	0	0
50,001-75,000	398	248	132	0	0	0
75,001-100,000	180	112	60	0	0	0
100,001-500,000	1,096	684	364	84	0	0
500,001-1,000,000	0	0	0	0	0	0
>1,000,000	0	0	0	0	0	0
Total	5,211	3,325	2,104	809	347	147
Lower Bound		2,841	1,632	533	210	86
Upper Bound		3,810	2,576	1,085	483	208

Note: Lower and upper bounds are based upon 95% confidence intervals. Numbers may not add to totals due to rounding. Subheadings indicate the NIRS system size strata.

Adapted from USEPA (1990a)

log-normally distributed with a geometric mean of 0.89 pCi/L and a geometric standard deviation of 2.68 pCi/L. Preliminary results are available from the National Residential Radon Survey (NRRS), which was designed to provide unbiased population estimates of radon exposure (USEPA 1991c). The arithmetic average of the measured radon concentration over all frequently occupied levels of a house was used as the measure of radon exposure for each of the 6,000 houses surveyed. The arithmetic mean concentration is 1.27 pCi/L and the geometric mean is 0.57 pCi/L with a geometric standard deviation of 5.1 pCi/L (USEPA 1991c).

The indoor air distribution of Nero et al. (1986) was used to estimate the relative contribution of radon released from water to indoor air concentration (USEPA 1990a). The arithmetic mean and distribution of transfer factors derived by Nazaroff et al. (1987) (mean of 8,930:1, standard deviation of 6,000:1) were applied to the distributions of water concentrations shown in Tables IV-3 and IV-4. For households served by groundwater supplies, radon in water contributes a mean of about 4% and a median of about 1% of total indoor radon. About 5% of households have a water contribution of 15% or more, but in these households, the total radon concentration is low (mean less than 1 pCi/L) (USEPA 1990a). Because the geometric mean air radon concentration found in the NRRS survey is lower than that of Nero et al. (1986) used in these calculations, a revision of this analysis using the results of the NRRS survey is likely to show a somewhat greater contribution to total indoor radon from radon released from water. For ingestion exposure, virtually no radon is found in food, and so drinking water is the entire source of ingested radon.

B. Other Sources

Elevated levels of radon in soil, water and ambient air can be produced by contamination from uranium mining or milling operations (USEPA 1990c). Disposal of waste from thorium or radium processing can also cause contamination of soil, groundwater and/or structures (USEPA 1990c). Such sources can result in high exposures to populations in the vicinity, particularly if homes are located on contaminated soil or made with contaminated materials, or if a contaminated aquifer is used as a source of potable water.

C. Assessment of Exposure Pathways

1. Ingestion Exposure

The extent of ingestion exposure to radon depends upon the amount of water that is consumed and the amount of radon actually in the water that is consumed. Radon is released from water at a rate that depends upon the surface area, agitation, temperature and duration of contact between the water and air (Becker and Lachajczyk 1984). Gesell and Prichard (1980) found that the loss of radon from water is slow at room temperature (about 0.15% per minute without agitation and about 20 times higher with agitation). Release is much more rapid at higher temperatures, and essentially all radon is released from water heated to the boiling point (Gesell and Prichard 1980). From these data, it is likely that essentially all radon is released from water used in cooking and to make hot beverages (coffee, tea, soup, etc.). Water used to prepare cold beverages from a concentrate or a dry mix could retain some radon if the beverage were prepared with minimal stirring and consumed shortly after preparation. However, total consumption of beverages prepared from concentrates and dry mixes less than 10% of the total consumption of water, coffee, tea and reconstituted soups (Pennington 1983), and no data are available to determine how quickly such beverages are consumed after preparation. As a simplification, it is assumed that the only ingested water that retains radon is water drawn directly from the tap ("direct tap water") (USEPA 1991b).

Available studies of tap water intake are based primarily on beverage intake data from the U.S. Department of Agriculture (USDA) 1977-1978 Nationwide Food Consumption Survey (USEPA 1991b). These studies are in general agreement that the intake of direct tap water averages about 0.6 to 0.7 L/day, and that about half of tap water is consumed as direct tap water (Pennington 1983, USEPA 1984, Ershow and Cantor 1989). This suggests that the best estimates of tap water consumption are about 0.7 L/day for direct tap water and 1.2 L/day for total tap water. For consistency with the practice of using 2 L/day for adult tap water consumption in the regulation of other drinking water contaminants, a value of 1 L/day for direct tap water is a reasonable, protective value, expressed to one significant figure (USEPA 1991b).

Some radon is lost even from fresh, cold tap water during the process of filling a glass and drinking from it (USEPA 1991b). A range of values for loss of radon from 0% to 53% have been reported for water during circumstances similar to the handling involved in consuming direct tap water (von Dobeln and Lindell 1964, Hursh et al. 1965, Suomela and Kahlos 1972, Partridge et al. 1979). A loss of 20% is considered a reasonable estimate of the typical loss during ingestion of direct tap water (USEPA 1991b).

2. Inhalation Exposure

Radon is released to household air from all uses of potable water, including cooking, showers, toilets, washing machines and dishwashers (USEPA 1991b). The influence of the water radon content on the air radon content of a house is evaluated in terms of a transfer factor. The transfer factor is the ratio of the concentration of radon in water (in pCi/L_w) to the concentration of radon in air (in pCi/L_a). The transfer factor depends upon the pattern of household water use (amount, timing, duration, agitation, temperature), the volume and air ventilation rate of the rooms in which water is used and the volume and air ventilation rate of the entire house (Becker and Lachajczyk 1984). The transfer factor has been measured directly, which is most easily done when water dominates as the source of indoor radon, and has been calculated based on measured ventilation rates, radon water-to-air transfer efficiencies, water uses and home volumes (USEPA 1991b).

Inhalation exposure to radon released from water supplies is influenced by a number of factors which vary from house to house and individual to individual. Two studies have used a transfer factor approach to analyze typical values and ranges for whole-house exposure to radon from water supplies. Becker and Lachajczyk (1984) suggested that a typical transfer factor is 14,000:1 and Nazaroff et al. (1987) derived a geometric mean transfer factor of 15,000:1 and an arithmetic mean transfer factor of 9,100:1. Actual transfer factors for American houses are expected to have a wide range, with most falling between 1,000:1 to 100,000:1 (Becker and Lachajczyk 1984, Nazaroff et al. 1987). Empirical assessments have generally supported a mean transfer factor close to 10,000:1 to 15,000:1, with a range of about two orders of magnitude (Gesell and Prichard 1980, AWWARF 1990, Hess et al. 1990).

The transfer factor approach may underestimate the concentration of radon gas to which individuals are exposed, because peaks of radon gas release from showers or other uses of water are not accounted for (Andelman 1990). USEPA (1991b) analyzed the extent of the underestimate of radon gas concentrations using an implementation of house-compartment models of McKone (1987) and Small et al. (1990). This analysis indicates that in a situation with a typical transfer factor, radon gas concentrations are underestimated by about 50%. The underprediction is less for individuals who remain only briefly in the bathroom after showering, who are away from home during peaks of water use such as laundry, who take baths rather than showers, who take short, cool or low-flow showers, whose showers or bathrooms are well ventilated, or who use more water than typical for uses other than showering. Conversely, the underprediction is greater for individuals who take long or high-flow showers or whose showers and bathrooms are poorly ventilated. In addition, when the conditions of water use, house size or house ventilation are such that the transfer factor is less than 15,000:1, elevated exposure during showers is less significant, and conversely, when conditions are such that the transfer factor is more than 15,000:1, elevated exposure during showers is more significant (USEPA 1991b).

The analysis of the difference in radon gas concentrations does not take into account the relative dosimetry of radon decay products in showers. Studies of the dosimetry of radon decay products have only addressed exposure conditions in mining environments or in general household air (see Section III.B.2). No data were located describing exposure conditions in a shower. However, it seems likely that the equilibrium factor, unattached fraction, aerosol size distribution and aerosol deposition characteristics would be different from those used to characterize the typical home or mining environment (USEPA 1991b). Limited experimental evidence confirms that the concentration of decay products builds up more slowly than the concentration of radon gas as radon is released during shower operation (McGregor and Gourgon 1980). It is possible that the dose in a shower would be much lower than in the general home environment, due to the low equilibrium factor, if the primary fate of decay products were to attach to the shower walls or to be scavenged by large, non-respirable drops of water. On the other hand, the low equilibrium factor could be somewhat counteracted by a high unattached fraction, if most decay products remained as ultrafine aerosols or became attached to mist particles that deposited with high efficiency in the respiratory tract. On balance, it appears that at a given radon gas concentration in pCi/L, the dose to the lung would be lower in a shower than in the general home environment, but more data and analyses are needed to confirm this. This remains as a substantial source of uncertainty in quantifying risk from radon released from household water supplies (USEPA 1991b).

For the purpose of deriving a relationship that quantifies the typical exposure of the United States population, a mean national transfer factor of 10,000:1 is used, selected near the lower end of the estimated range of typical values (9,100:1 to 15,000:1) (Becker and Lachajczyk 1984, Nazaroff et al. 1987) to account for increased exposure during showering (USEPA 1991b).

D. Summary

The occurrence of radon in ground water supplies follows an approximately log-normal distribution, with a mean value of about 650 pCi/L (Longtin 1988, USEPA 1988). These data can be used to estimate population exposure from community water supplies and can be extrapolated to estimate exposure from nontransient non-community water supplies (USEPA 1990a). Approximately 800,000 people are exposed to water containing more than 2,000 pCi/L of radon, 650,000 from community water supplies and 150,000 from nontransient non-community water supplies (USEPA 1990a). Approximately 17,000,000 people in the United States are exposed to more than 300 pCi/L in community and nontransient non-community water supplies (USEPA 1991a). Another source of radon exposure is the accumulation of radon from soil gas in indoor air. Radon from water supplies contributes a mean of about 5% of the indoor air radon (USEPA 1990a). Radon from water supplies is the only source of ingestion exposure to radon.

Human exposure to radon in water supplies occurs due to ingestion of radon-containing water and inhalation of radon released from water from household uses such as showers, washing machines, etc. Ingestion exposure is evaluated based on consumption of 1 L/day of direct tap water, which loses 20% of its initial radon content during the processes of filling a glass and drinking the water (USEPA 1991a). Inhalation exposure is evaluated in terms of a water-to-air transfer factor. The transfer factor is the ratio of the water concentration of radon to the average increase in the air concentration of radon attributable to release from water. The national average transfer factor is estimated to be about 10,000:1 to 15,000:1, and to range from about 1,000:1 to 100,000:1 (Becker and Lachajczyk 1984, Nazaroff et al. 1987). The transfer factor approach may underestimate exposure to radon gas because it does not account for peaks of concentration from activities such as showering. The extent of the underprediction of radon gas exposure is typically about 50% (USEPA 1991a). The extent of the underprediction of exposure to radon decay products is likely to be less because of the lower equilibrium factor during showers. However, the dosimetry of radon decay products under showering conditions has not been investigated, and this remains as a substantial source of uncertainty in evaluating inhalation exposure (USEPA 1991a). Use of a transfer factor of 10,000:1 provides a reasonable estimate of typical human exposure to radon and radon decay products released from all household uses of water (USEPA 1991a).

Quantitative uncertainties in evaluation of human exposure to radon in potable water supplies are discussed in Chapter IX.

V. HEALTH EFFECTS IN ANIMALS

No studies were located describing health effects in experimental animals exposed to radon by the ingestion route. Studies of effects observed in animals exposed to radon and decay products by inhalation are discussed in this chapter.

A. Short-Term Exposure

Morken (1955) exposed mice to 220,000,000 pCi/L of radon in air for 5 to 40 hours. Concentrations of decay products were very low. The 30-day LC_{50} for radon in mice was estimated to be a one-hour exposure to between 5,700,000 and 8,800,000 pCi/L. Observed toxic effects included emaciation, reddening of the ears, pseudoparalysis and anemia.

B. Longer-Term Exposure

No data were located concerning noncancer health effects of radon or radon decay product exposures of 90 days or more in experimental animals.

C. Reproductive and Developmental Effects

Leonard et al. (1981) exposed male and female mice to high natural background levels of radiation from gamma rays and radon. Gamma exposure was 8 mrad/hr and radon exposure was 4 WLM over 4 months. Fertility of each sex was assessed for 6 months after exposure by mating with unexposed controls. The reproductive success, measured by number of offspring weaned, was significantly higher for exposed males and significantly lower for exposed females, compared to controls. The authors attributed the fertility effects to gamma irradiation of gonads, but no experiments were done with mice exposed only to radon and radon decay products to verify this conclusion.

D. Mutagenicity

In general, ionizing radiations, including alpha particles, are known to cause mutations and chromosomal aberrations in mammalian cells (NAS 1988). Two studies discussed below have specifically examined the genotoxicity of radon inhalation in animals.

Leonard et al. (1981) exposed rabbits to radon in air either at high natural background levels (28 WLM over 28 months) or under controlled conditions (10.7 WLM over 1 month). Chromosomal aberrations in lymphocytes were induced under natural background but not under controlled exposures, indicating that gamma irradiation from sources other than radon was the main cause of the chromosomal aberrations.

Sperlich et al. (1967) exposed male Drosophila to an atmosphere containing radon and decay products and investigated the frequency of sex-linked lethal mutations. No increased mutation rate was found at exposures to 2,800,000 pCi/L for 24 hours or to 3,500 pCi/L for 21 days, but an increased rate was found at an exposure of 78,600,000 pCi/L for 24 hours.

E. Carcinogenicity

Inhalation exposure of experimental animals to radon and radon decay products has been shown to cause lung cancer in numerous experiments.

Selected recent studies are summarized in this section. Additional studies have recently been reviewed (NAS 1988, ATSDR 1989).

Chameaud et al. (1982) exposed groups of 28 to 50 male Sprague-Dawley rats for 3 to 6 hours per day for 2.5 to 8 weeks to total cumulative exposures of 100, 500 or 4,000 WLM. Lung cancer incidence was determined by histologic examination of lungs one year after radon exposure. The incidences in the 100, 500 and 4,000 WLM groups were 0%, 7% and 34%, respectively. Rats similarly exposed to radon and then exposed to cigarette smoke for one year had a significantly increased incidence of lung cancers, and the tumors were more invasive.

Chameaud et al. (1984) exposed groups of 500 to 600 male rats twice a week in 1-hour sessions for 21 or 41 weeks to total cumulative exposures of 20 or 40 WLM. Animals were examined after natural lifespan, up to 31 months after beginning of exposure. Lung cancer incidence was 5/600 in controls, 11/496 at 20 WLM and 19/497 at 40 WLM. The tumors in the exposed rats were of earlier onset, larger size and greater invasiveness than in controls. The authors concluded that the dose-response relationship was linear or supralinear, and that exposure to 20 WLM doubled the spontaneous lung cancer rate.

Cross et al. (1981) exposed 102 male Syrian Golden hamsters for 6 hours per day for up to 600 days to 690 WL of radon decay products. The incidence of non-pulmonary neoplasms was no different from controls. Lung tumors developed in only three animals and only in animals exposed to more than 8,000 WLM. The authors suggested that the hamster is resistant to lung cancers and may not be a good model for human pulmonary carcinogenicity.

Cross et al. (1982) exposed male and female beagle dogs for 4 hours per day for up to 4.5 years to 105,000 pCi/L radon, 605 WL of radon decay products and 13 mg/m^3 uranium ore dust (mean diameter 0.6 to 2.1 μm). Respiratory tract tumors developed in 8/20 dogs, all of which had cumulative exposures greater than 13,000 WLM. Significantly fewer lung tumors developed among dogs in which cigarette smoke exposure alternated with radon exposure, possibly due to decreased dose to sensitive cells from physiological changes caused by cigarette smoke.

Cross et al. (1984) exposed groups of 32 to 48 male rats for 6 hours per day for up to 600 days to 640 or 2,560 WL of radon decay products and 22 mg/m^3 uranium ore dust (mean diameter about 1 μm) at various exposure rates and attachment fractions. Significantly increased rates of lung tumors were found among all exposed groups. The risk of lung tumors increased with decreased exposure rate, but the trend was not statistically significant. The risk of lung cancer increased with increased unattached fraction and decreased equilibrium factor, and the trends were of borderline statistical significance.

A review of these and other studies indicates that the risk of lung cancer in experimental animals exposed to radon and radon decay products is influenced by cumulative exposure, exposure rate, unattached fraction and equilibrium factor (Cross et al. 1984, NAS 1988). Risk increases essentially linearly with increasing cumulative exposure up to levels causing lifespan shortening, with no evidence of a threshold down to 20 WLM. Risk decreases at higher exposure rates (about a 3-fold difference between 50 and 500 WLM/week). Risk increases with unattached fraction (about a 50% increase at 10% over 2%

unattached fraction). Risk decreases with increasing equilibrium factor (about a 30% decrease at 40% over 10% equilibrium).

F. Summary

No information was located concerning health effects in animals of ingestion of radon. Prolonged inhalation exposure of rats or dogs to radon and radon decay products causes lung cancer. Hamsters appear to be resistant to the carcinogenic effects of radon. No threshold for lung cancer induction by radon exposure is apparent in animal experiments. Quantitative analyses indicate that risk increases with increasing total exposure, with decreasing exposure rate, with increasing unattached fraction and with decreasing equilibrium factor (NAS 1988).

VI. HEALTH EFFECTS IN HUMANS

A. Clinical Case Studies

No information was located concerning clinical case studies of humans exposed to radon by ingestion. Miners working in metal mines in the Erz Mountains of eastern Europe were recognized as having an increased mortality from respiratory disease as early as the sixteenth century (Agricola 1556). Harting and Hesse (1879) described clinical and autopsy findings establishing lung cancer as the cause of death known as "miner's disease". By the early part of the twentieth century, exposure to radioactivity was identified as the cause of the lung cancers among these miners (Pirchan and Sikl 1932, Teleky 1937). Subsequent epidemiologic studies of these and other cohorts have been carried out, as discussed in the next section.

B. Epidemiologic Studies

1. Effects of Radon in Drinking Water

Five studies were located describing health effects in populations exposed to radon in drinking water.

Stenstrand et al. (1979) found a significant increase in chromosomal aberrations in peripheral blood lymphocytes of 18 Finns exposed to radon concentrations in water from 2,900 to 1,200,000 pCi/L compared to 9 unexposed controls. The authors were not able to determine a dose-response relationship, but found that the incidence of chromosomal aberrations increased with age and length of exposure. Exposure to radon included both ingestion and inhalation in the exposed individuals.

Hess et al. (1983) investigated the association between radon concentration in drinking water and cancer mortality in 16 Maine counties. Water radon concentration was estimated based on measurements of water samples from more than 2,000 public water supplies. Lung cancer mortality rates were obtained for each county from 1950 to 1969. There was a significant positive correlation between estimated county average radon concentrations and mortality from lung cancer and from all cancers combined. Marginally significant associations were found with prostate, connective tissue and brain plus central nervous system cancers.

Collman et al. (1988) investigated the association between radon concentration in drinking water and cancer mortality in North Carolina counties. Water radon concentrations were estimated based on measured values combined with geologic data. State vital statistics records were used to determine the causes of death between 1978 and 1982. Counties having radon levels in the lowest tertile (0 to 228 pCi/L) were considered unexposed and the rest of the counties were considered exposed (radon levels between 229 and 10,892 pCi/L). A total of 783,282 persons were unexposed and 3,198,170 were exposed. No significant increase was found among exposed persons in mortality from upper respiratory cancer, gastrointestinal cancer, bone cancer, breast cancer or leukemias. Smoking, occupational exposure to carcinogens, migration and cancer survival were not controlled for.

Mose et al. (1990) investigated correlations between measured radon water levels and self-reported family health among 1,400 residents of southern

Maryland and northern Virginia. Among nonsmokers and former smokers, self-reported rates of all types of cancer were increased among residents with radon concentrations above 2,500 pCi/L in water, but rates were only roughly age-adjusted and no statistical analyses were performed to determine whether any observed effects were significant.

Collman et al. (1991) investigated childhood cancer mortality in the same North Carolina counties studied previously (Collman et al. 1988). Age- and sex-adjusted cancer death rates from 1950 to 1979 among children under age 15 were calculated for counties divided into tertiles on the basis of geometric mean measured radon concentration of groundwater supplies. Death rates for all childhood cancers combined and for childhood leukemias showed a significant increase in medium and high radon counties with a dose-response gradient. For leukemias, compared to counties with less than 229 pCi/L of radon in water, the relative risk in counties with 229 to 1,375 pCi/L was 1.26 (95% confidence interval 1.08-1.47) and the relative risk in counties with 1,376 to 10,692 pCi/L was 1.33 (95% confidence interval 1.13-1.57). Collman et al. (1991) noted that mean county radon concentrations are only a rough measure of exposure and that confounding factors or biases may have influenced the results.

2. Effects of Radon and Radon Decay Products in Air

A large number of studies have investigated the health effects of inhaled radon and radon decay products. Several of the major recent studies of underground miners are summarized below. In addition to the summarized analyses, several other studies, recently reviewed by the BEIR IV committee

(NAS 1988), have established an increased risk of lung cancer among radon-exposed miners.

Mutagenicity

Brandom et al. (1978) found an increased incidence of chromosomal aberrations in peripheral blood lymphocytes among Colorado Plateau uranium miners compared to matched controls. Miners were divided into five exposure groups, and a clear dose-response relationship was observed in the lowest four groups, with almost a six-fold increase in total aberrations over controls. In the highest exposure group (above 3,000 WLM), the frequency of aberrations declined. The authors suggested that the observed chromosomal aberrations provided an indication of radiation-induced health risk among these miners.

Pohl-Ruling and Fischer (1979) found an increased frequency of chromosomal aberrations in peripheral blood lymphocytes of workers exposed to 3,000 pCi/L of radon for 2 to 4 hours per day in a former gold mine now used for "radiation therapy".

Tuschl et al. (1980) found an increase in DNA repair in peripheral blood lymphocytes in the same population of workers. The DNA repair rate was higher in juvenile lymphocytes than in fully-differentiated lymphocytes. The increased DNA repair rates may reflect induction of repair enzymes in response to DNA damage.

Reproductive and Developmental Effects

Wiese (1981) investigated reproductive effects possibly associated with prevalence of uranium mining in the Four Corners areas of New Mexico, Arizona and Utah. Suggestive associations were found with rates of congenital malformations and spontaneous abortions, changes in the ratio of male to female live births and changes in the percent Y bodies in sperm of uranium miners. However, none of these data were considered conclusive.

Wiese and Skipper (1986) followed up on the previous study by surveying reproductive outcomes of 486 male uranium miners and 213 male potash miners. Reported birth outcomes were compared for conceptions occurring before or after the start of underground mining, and also before or after 1970, since radon exposure decreased substantially in the late 1960's. A statistically significant decrease was found in the mean birth weights of offspring conceived after the start of underground work, but only among men beginning mining after 1970. No significant or consistent trends were found in frequency of low birth weights, sex ratio, miscarriages or infertility.

Cancer

Hornung and Meinhardt (1987) analyzed Colorado Plateau uranium miners. Data on this cohort had been gathered by the U.S. Public Health Service, combining an industrial-hygiene survey of radon decay product concentrations in WL with a medical study of miners and millers. Exposure estimates were based on yearly average measured WL concentrations, interpolated and extrapolated over time and between mines, and on individual work histories. Information on smoking habits was obtained by surveys, and mortality and causes of death were defined from death certificates. Hornung and Meinhardt (1987) considered a cohort of 3,346 white male miners with a mean exposure of 821 WLM and a mean follow-up time of 22 years (from 1950 through December 31, 1982). A total of 256 deaths from lung cancer were observed in this cohort, with a clear dose-response trend with increasing exposure that was linear below 600 WLM. Significant increased risk of lung cancer was found at exposures above 120 WLM. Risk was increased at older ages of first exposure, lower rates of exposure, shorter time since last exposure and habit of cigarette smoking. Samet and Horning (1990) considered the major limitation of this cohort to be the high and somewhat uncertain exposures received by most members.

Sevc et al. (1988) recently analyzed Czechoslovakian uranium miners. Exposure estimates were based on measured concentrations of radon gas and decay products and on individual work histories determined from payroll cards. Lung cancer mortality was based on information from health facilities. Detailed information on smoking was not available, but based on a random sample, about 70% of the workers were smokers. Sevc et al. (1988) analyzed four cohorts of 1,561 to 3,799 workers, with mean exposures of 3.2 to 303 WLM and mean follow-up times of 6 to 30 years. A total of 488 lung cancers were observed in these cohorts, with a clear dose-response trend. Elevated risk of lung cancer was significant at exposures as low as 50 WLM. The cohorts of current miners with low total exposures also had short follow-up times, but preliminary results indicated that attributable risk was linear down to doses not far above background. Risk was increased at older ages of first exposure and lower rates of exposure. Samet and Hornung (1990) considered the major limitations of this cohort to be the use of attributable risk rather than relative risk and the lack of radon decay product measurements prior to 1960.

Muller et al. (1985) analyzed Ontario uranium miners. Exposure estimates were based on company WLM records after 1968 and on measured and extrapolated WL values and work histories prior to 1968. Lung cancer mortality was determined through death certificates identified from national mortality data bases. No information on smoking was available. Miners with asbestos exposure or experience in mining other metals were considered as separate cohorts. Muller et al. (1985) analyzed 15,984 uranium miners, with mean exposures in the range of 40 to 90 WLM and a mean follow-up time of 15 years. A total of 119 lung cancers were observed in this cohort, with a clear dose-response trend. Significantly increased risk was found above exposures of 40 WLM. Risk decreased with increasing age. Samet and Hornung (1990) considered the major limitations of this study to be lack of information about smoking and the relatively young age of the cohort.

Radford and Renard (1984) analyzed Malmberget iron miners. Exposure estimates were based on assuming that groundwater was the source of radon and that the concentration of radon in groundwater and ventilation conditions were approximately constant from 1915 to 1972. Work histories were determined from company and union records. Lung cancer mortality was obtained from death certificates of workers traced from parish records. Information on smoking was available for each worker dying from lung cancer and for selected workers surveyed in 1972 to 1977. Radford and Renard (1984) analyzed 1,415 men, with mean exposures of 81 WLM and a mean follow-up time of 44 years. A total of 50 lung cancers were observed in this cohort, with a clear dose-response trend. Significantly increased risk was found in the lowest exposure group, with a mean exposure of 27 WLM. Samet and Hornung (1990) considered the major limitations of this study to be the methods used to assign exposure assessment and the incomplete smoking histories.

Howe et al. (1986) analyzed Eldorado (Saskatchewan) uranium miners. Exposure estimates were based on measured and extrapolated WL values and work history determined from employment records. Lung cancer mortality was determined by linkage with a national mortality data base. No information on smoking was available. Howe et al. (1986) analyzed 8,487 workers, with mean exposures of 20 WLM and a mean follow-up time of 14 years. A total of 65 lung cancers were observed in this cohort, with a clear dose-response trend. Significantly increased risk was found at exposures above 100 WLM. The major limitations of this study are the interpolation of median WL concentrations, the short follow-up times and the lack of smoking histories.

Morrison et al. (1988) recently analyzed Newfoundland fluorospar miners. The source of radon in the mines was seepage of radon-containing groundwater. Exposure estimates were based on back-extrapolation of WL measurements in one mine in 1959 and on occupational histories. Lung cancer mortality was ascertained by linkage with a nationwide mortality data base and through other records. Smoking histories were available for 48% of the population. Morrison et al. (1988) analyzed 1,772 workers, with a mean exposure of 380 WLM followed from 1950 to 1984. A total of 113 lung cancer deaths were observed in this cohort, with a clear dose-response trend. Significantly increased risk was found at exposures above 100 WLM. Highest relative risks were found among those first exposed between ages 15 and 19. The major limitations of this study are incomplete smoking histories and the uncertainty in exposure estimates.

Samet et al. (1989) recently analyzed New Mexico uranium miners in a case-control study. Exposure estimates were based on interpolation and extrapolation of WL measurements and on work histories derived from company records, medical histories and surveys. Lung cancer mortality was determined by mortality searches, review of a population-based cancer registry and information from physicians and mining companies. Information on smoking was available for nearly all subjects. Samet et al. (1989) analyzed 3,500 workers, with mean exposures of 113 WLM and a mean follow-up time of 18 years. A total of 65 cases of lung cancer were observed in this group, and a case-control analysis indicated a clear dose-response trend. Odds ratios increased with increasing cumulative exposure, using 0 to 99 WLM as the reference category. Risk increased with decreasing age at first exposure and with time since exposure. A multiplicative interaction between radon exposure and cigarette smoking was found. Samet and Hornung (1990) considered the major limitation of this study to be that it is a case-control rather than a cohort study.

Lubin et al. (1990) recently analyzed Chinese tin miners in a case-control study. Exposure estimates were based on WL measurements in current or recreated mines and on lifetime work history obtained by interview. Lung cancer mortality was determined from cases reported to the cancer registry of the Yunnan Tin Corporation. Information on smoking was obtained by interview. Lubin et al. (1990) analyzed 74 cases and 74 exposed controls, with mean exposures of 380 WLM. A clear dose-response trend was observed in this analysis, with an increased relative risk in the lowest exposure category, 1 to 99 WLM. Excess relative risk decreased with time since exposure and with increasing exposure rate. Workers first exposed before age 13 had a non-significantly lower risk than workers first exposed after age 13, but the joint effect of age at exposure and time since exposure was not analyzed. Risk increased with tobacco smoking by water-pipe in a manner between an additive and multiplicative interaction. The major limitations of this study are the use of the case-control rather than cohort design and the uncertainty in the exposure estimates.

In addition to studies of underground miners, several descriptive and epidemiologic studies have investigated the association between lung cancer incidence and exposure to radon in the home. In 1989, Samet (1989) reviewed the principal published reports of investigations of the general population. Of the 11 descriptive studies reviewed, 5 found significant associations between excess risk of lung cancer and radon exposure, 2 found nonsignificant associations, 3 found no association and 1 found a negative association (Samet 1989). Of the 10 case-control or cohort studies reviewed, 7 found an association between lung cancer risk and radon exposure and 3 found no association (Samet 1989). Cohen (1990) recently published an expanded version of the descriptive study reviewed by Samet (1989) that found a negative association between geometric mean radon levels in a county and the age-adjusted lung cancer mortality rate in that county, and suggested that this unexpected result required further study and interpretation. Svensson et al. (1989) recently reported an additional case-control study of 210 female lung cancer patients and matched controls in Stockholm, Sweden that found a significant association between lung cancer and estimated cumulative indoor radon exposure. Schoenberg et al. (1990) recently reported the results of an additional case-control study of 433 female lung cancer patients and 402 matched controls in New Jersey. A trend of increasing risk with increasing estimated cumulative radon exposure was found. The increase was significant at the 9% level and was of a magnitude consistent with that found in miner studies (Schoenberg et al. 1990).

Descriptive and epidemiologic studies of indoor radon exposure have often used small numbers of subjects, and many have been hampered by the difficulty in establishing valid individual exposure estimates (Samet 1989). Overall, available studies present results suggestive of a possible relationship between indoor radon exposure and lung cancer (NAS 1988, Lubin and Boice 1989, Samet 1989).

C. High-Risk Populations

Direct information is not available to establish whether children or women may be more or less sensitive to the effects of radon and radon decay products than the male miners in whom lung cancer has clearly been established as the result of exposure to radon and radon decay products in air. Risk in miners appears to be proportional to age-specific and smoking-specific lung cancer rates, which implies that risk may also be proportional to sex-specific risk as well (NAS 1988). The only known population of females exposed to high indoor concentrations of radon and radon decay products are women who worked in radium dial painting facilities in the early part of this century (Stebbings et al. 1984). Rates of lung cancer were elevated to about the degree expected based on estimated exposure using risk estimates derived from miner data (Stebbings et al. 1984). However, the excess in lung cancer was not significant, and less than 10% of the cohort was exposed to more than 30 WLM (Stebbings et al. 1984).

The concern about the increased sensitivity in children is the issue of whether exposure during childhood will increase the lifetime risk of cancer to a greater degree than an equal exposure during adulthood. The dose to sensitive lung cells from a given WL concentration may be higher in children than in adults due to differences in bronchial morphometry, fraction of particles removed in the mouth and nose, mucociliary clearance, tidal volume of breathing and breathing frequency (Samet 1989). The relative risk of lung cancers and other cancers induced by gamma ray exposure is higher in atomic bomb survivors irradiated as children (Preston et al. 1987), but it is not known if the elevated relative risks will persist throughout a lifetime (NAS 1990). As discussed in the previous section, the study of Lubin et al. (1990) indicated no difference in relative risk of lung cancer between Chinese tin miners first exposed to radon before or after age 13. Based on the increased relative risk among atomic bomb survivors and the increased dose per unit exposure, the ICRP 50 committee recommended a risk coefficient that is 3 times higher for exposures under age 20 than for adults (ICRP 1987). In contrast, the BEIR IV committee acknowledged these same considerations, but recommended a model that assumed that children were not more sensitive than adults, noting that projections of risk from lifetime exposure in their preferred model would not be much affected by assumptions about childhood sensitivity (NAS 1988).

Cigarette smokers appear to be more sensitive than nonsmokers to lung cancer induced by inhalation of radon decay products. The BEIR IV committee analyzed the interaction between smoking and exposure to radon decay products and concluded that the data are consistent with a multiplicative but not an additive interaction (NAS 1988). Subsequent studies have supported this conclusion (Samet et al. 1989, Lubin et al. 1990). A multiplicative interaction implies that the risk from inhalation of radon decay products is proportional to the increased risk of lung cancer induced by smoking. This can be expressed as a risk multiplier, relative to the risk derived for the average member of the population of unspecified smoking status (USEPA 1990b).

Table VI-1 presents the risk multiplier for nonsmokers, former smokers and current smokers (USEPA 1990b).

D. Summary

Epidemiologic studies have clearly established that inhalation exposure to radon and radon decay products cause lung cancer in miners. Dose-response relationships are generally linear, with increased risk of lung cancer often observed even in the lowest exposure groups, although the increases are not always significant in the lowest groups (NAS 1988). Increased risk of lung cancer among populations exposed to radon and radon decay products in home air has been observed in several studies (Samet 1989). Studies examining incidences of other types of cancer among populations exposed to radon in drinking water have found mixed results (Hess et al. 1983, Collman et al. 1988, 1991). Children may be at higher risk of developing lung cancer later in life than adults exposed to equal concentrations of radon decay products (ICRP 1987). Smokers are at substantially higher risk than nonsmokers for developing lung cancer from exposure to radon decay products (NAS 1988).

Table VI-1 Relative Risks of Lung Cancer Induced by Inhalation of Radon Decay Products For Different Smoking Categories

Smoking Category	Risk Multiplier (relative to population average)
Nonsmoker	0.2
Former smoker	0.8
Current smoker	
Average	1.8
<10 cigarettes/day	0.7
10-20 cigarettes/day	1.8
21-39 cigarettes/day	3.2
≥40 cigarettes/day	4.5

Source: USEPA 1990b

VII. MECHANISM OF TOXICITY

A. Effects of Ionizing Energy

The potential for ionizing radiation to cause tissue damage and cancer was established within 15 years after the discovery of X-rays (USEPA 1989b, Upton 1975). Extensive studies have been done on radiation effects in man and animals following exposure to X-rays, gamma rays, radium, radon, thorium and other sources of ionizing radiations.

Ionizing radiation is not an organ- or tissue-specific carcinogen. Storer (1975) stated: "Ionizing radiation in sufficiently high dosage acts as a complete carcinogen in that it serves as both initiator and promoter. Further, cancers can be induced in nearly any tissue or organ of man or experimental animals by the proper choice of radiation dose and exposure schedule." The following tissues have shown the induction of cancers in man by radiation in one or more epidemiological studies: thyroid, female breast, lung, bone marrow (leukemia, multiple myeloma), stomach, liver, large intestine, small intestine, brain, salivary glands, bone, esophagus, urinary bladder, pancreas, rectum, lymphatic tissues, skin, pharynx, uterus, ovary, mucosa of cranial sinuses and kidney (USEPA 1989b).

The damage caused by ionizing radiation involves a number of rapid radiochemical reactions. Absorption of energy from alpha and beta particles induces ions and excited radicals in matter, including aqueous solutions. Absorption of energy from x- and gamma-radiations by atoms results in the ejection of electrons from the atoms. These ejected high-speed electrons then produce ionizations in the same manner as the alpha and beta particles. These types of interactions occur in about the first 10^{-14} seconds (Bacq and Alexander 1961, Andrews 1961).

The ions and excited radicals induced in cells (aqueous solutions) occur both in organic molecules of the cell contents and in the water surrounding them. In addition, specific compounds, such as H_2O_2, and radicals, such as $HO_2^{\cdot}$, are formed in the water phase. Further chemical interactions can then occur with proteins, sugars, lipids, enzymes and more importantly, DNA and RNA. Types of damage to DNA and other organic molecules include bond scission, chain scission, cross-linking, single and double strand breaks and development of adducts. These interactions are generally complete by about 10^{-4} seconds after initial exposure (Bacq and Alexander 1961, Andrews 1961).

Ionizing radiation delivered at sufficiently high dose and dose rate leads to cellular death and eventually death of a living organism. At lower doses and dose rates ionizing radiations induce cancer. The ionizing radiations can inhibit mitosis and cause chromosome aberrations including nondisjunction, clastogenesis and point mutations (Bacq and Alexander 1961, Andrews 1961). These effects may be demonstrable minutes to years after exposure.

While carcinogenesis and mutagenesis has not been investigated for every radionuclide, the results of studies that have been performed are consistent with expected effects of ionizing radiation. Mammalian cells in culture have been transformed; chromosome aberrations have been observed in cultured peripheral lymphocytes; even activation, by frame shift due to a single base deletion in an oncogene, has been reported (USEPA 1989b). Induction of enzymes of unscheduled DNA repair, perhaps signaling error-prone repair, has also been noted (Tuschl et al. 1980, 1983, Olivieri et al. 1984).

Continuing epidemiological studies have shown increased cancer mortality in persons exposed to X-rays, gamma rays and various internal emitters such as radon, radium and thorium, at various ages (including *in utero*) (USEPA 1989b). Additional support comes from the studies of mice, rats, hamsters, guinea pigs, cats, dogs, sheep, cattle, pigs and monkeys that have demonstrated increased incidence of cancer following exposure to a source of ionizing radiation (USEPA 1989b).

B. Radionuclide Dosimetry

1. Ingestion Exposure

The concentration of radon in various organs following ingestion exposure is calculated using a toxicokinetic model developed by Crawford-Brown (1990) (see Section III.A). These concentrations were used to calculate dose to the organs in that model (see Figure III-1).

Normalizing to intake of 1 pCi of radon, the concentration of radon in organ (i) as a function of time in seconds (t) after ingestion is designated $C_i(t)$. The units of $C_i(t)$ are pCi/g (or pCi/cm^3, assuming an organ density of 1 g/cm^3). Crawford-Brown (1990) calculated integral organ burden, B_i in decays/g, from the following equation:

$$B_i = \int_0^t 0.037 \text{ (decays/sec)/pCi} \times C_i(t)dt \qquad \text{VII-1}$$

The radiation dose to an organ, D_i in rad, is calculated from the following equation:

$$D_i = B_i \times 19.2 \text{ MeV/decay} \times 1.6\text{x}10^{-8} \text{ rad/(MeV/g)} \qquad \text{VII-2}$$

The factor for the total alpha energy per decay is the sum of the alpha decay energies of Rn-222 (5.5 MeV), Po-218 (6.0 MeV) and Po-214 (7.7 MeV), based on the assumption that all decay products decay at the site they are produced. If only the first two alpha decays take place before removal, the dose is reduced by a factor of 0.60. Crawford-Brown (1990) included in the dose to the lung a component from the decay of radon in alveolar sacs prior to exhalation. This increases the dose by about 25% over than calculated only from decay in tissue.

The absorbed dose rate for each target organ is calculated using equations VII-1 and VII-2. The output from these calculations is a list of organs with doses of high-LET radiation at various ages corresponding to ingestion of 1 pCi of radon. These calculated organ-specific doses are combined with risk coefficients derived from quantitative evaluation of human cancer risk from various types of ionizing radiations (see Section VIII.B) to estimate organ-specific and total risk corresponding to radon ingestion. Uncertainties in these dosimetric calculations are discussed in Chapter IX.

2. Inhalation Exposure

Estimation of risk from inhaled radon decay products is based on extrapolation of lung cancer rates among underground miners with measured exposures, with no intervening calculation of absorbed dose (USEPA 1989b).

Although dosimetric models for inhaled radon decay products are not used for absolute prediction of risks, they are used to evaluate quantitative extrapolation between exposure in mines and in homes (see Section III.B.2).

The dosimetric portion of the RADRISK model is used in the calculation of risks from inhalation of radon gas. Ionizing radiation is known to induce cancer in many organs and tissues, and so risk estimates for internally deposited radon and radon decay products are based on the distribution of the radionuclides in tissues and the ionizing radiation dose associated with that distribution. The toxicokinetic model used to estimate distribution is described in Section III.B.1. As explained there, the radionuclide concentration in various organs is calculated for a lifetime inhalation of 1 pCi of radon per year. For all organs or tissues where there is no organ-specific model, the activity in the organ compartment "other" is considered to be uniformly distributed in all body tissues not specified.

The rate at which radiation is absorbed in a given organ depends on the concentration of each radioactive element in that organ and in surrounding organs, the frequency with which each element decays, the energy of each decay event and the fraction of emitted energy that is absorbed in the particular organ (Sullivan et al. 1981). The concentrations of radionuclides resulting from radon gas inhalation are calculated using the biokinetic model described in Section III.B.1. The frequency and energy of the radionuclide decay events are well-known physical properties. For alpha and beta particles the specific absorbed fraction (see below) is assumed to be inversely proportional to the mass of a given target organ; cross irradiation from sources outside the target tissue is assumed to be negligible except for source tissues in close proximity (USEPA 1986). [e.g.; sources, skeletal tissue - target, active bone marrow and endosteal cells (Thorne, 1977, ICRP 1980)]. Absorption of photons (i.e., gamma irradiation) is calculated by the Monte Carlo method (USEPA 1989b).

The equation used to calculate the rate of radiation absorption from element i in organ X is:

$$\dot{D}_i(X;t) = \sum_{k=1}^{M} A_{ik}(t)\ S_i(X \leftarrow Y_k) \qquad \text{(VII-3)}$$

where:

$\dot{D}_i(X;t)$ = the absorbed dose rate to organ X at time t due to radionuclide i in source organs $Y_1, Y_2, \ldots Y_M$. The first source organ Y_1 is organ X.

M = the total number of source organs contributing irradiation to organ X.

$A_{ik}(t)$ = the activity at time t of radionuclide i in source organ Y_k.

$S_i(X \leftarrow Y_k)$ = the dosimetric source (S) factor for irradiation of organ X by source organ Y_k.

The S factor is calculated by the following equation:

$$S_i(X \leftarrow Y_k) = c \sum_{m=1}^{N} f_m\ E_m\ \phi_m(X \leftarrow Y_k) \qquad \text{(VII-4)}$$

where:

c = a constant that depends on units of dose, energy and time being used.

N = the total number of decay events m (e.g., alpha decay, beta decay).

f_m = intensity of decay event m (number per disintegration).

E_m = average energy of decay event m in Mev.

$\phi_m(X \leftarrow Y_k)$ = specific absorbed fraction, ie., the fraction of emitted energy from source organ Y_k absorbed by target organ X per gram of tissue in organ X.

The S factor is similar to the SEE factor used by the ICRP to account for cross irradiation terms in ICRP Publication 30. However, the SEE factor includes a quality factor for high-LET radiation whereas the RADRISK calculations carry forward high-LET and low-LET dose rates as separate entities (USEPA 1989b, Dunning et al. 1984).

The absorbed dose rate for each target organ is calculated by summing equation VII-3 over all radionuclides i. The output from these calculations is a list of organs with doses of high-LET and low-LET radiation at various ages corresponding to inhalation of 1 pCi/year of radon. The RADRISK model combines these calculated organ-specific doses with risk coefficients derived from quantitative evaluation of human cancer risk from various types of ionizing radiations (see Section VIII.B) to estimate organ-specific and total risk corresponding to inhalation of radon gas. Uncertainties in dosimetric calculations are discussed in Chapter IX.

C. Summary

The harmful effects of ionizing radiation are believed to be the result of damage to DNA and other cellular components caused by chemical reaction with ionized molecules, particularly active oxygen species. Inhalation of radon decay products results primarily in radiation exposure to the lung (NAS 1988). The dose of radiation resulting from exposure to radon gas by ingestion or inhalation varies from organ to organ. Models have been developed to calculate the rate at which radiation is absorbed in a given organ, based on the concentration of radioactive elements in that organ and in surrounding organs, the frequency with which each element decays, the energy of each decay event and the fraction of emitted energy that is absorbed in the particular organ (USEPA 1989b, Crawford-Brown 1990). Uncertainties in dosimetric calculations are discussed in Chapter IX.

where:

c = a constant that depends on units of dose, energy and time being used.

N = the total number of decay events m (e.g., alpha decay, beta decay).

f_m = intensity of decay event m (number per disintegration).

E_m = average energy of decay event m in Mev.

$\phi_m(X \leftarrow Y_k)$ = specific absorbed fraction, ie., the fraction of emitted energy from source organ Y_k absorbed by target organ X per gram of tissue in organ X.

The S factor is similar to the SEE factor used by the ICRP to account for cross irradiation terms in ICRP Publication 30. However, the SEE factor includes a quality factor for high-LET radiation whereas the RADRISK calculations carry forward high-LET and low-LET dose rates as separate entities (USEPA 1989b, Dunning et al. 1984).

The absorbed dose rate for each target organ is calculated by summing equation VII-3 over all radionuclides i. The output from these calculations is a list of organs with doses of high-LET and low-LET radiation at various ages corresponding to inhalation of 1 pCi/year of radon. The RADRISK model combines these calculated organ-specific doses with risk coefficients derived from quantitative evaluation of human cancer risk from various types of ionizing radiations (see Section VIII.B) to estimate organ-specific and total

VIII. QUANTIFICATION OF TOXICOLOGICAL EFFECTS

General Approach for Drinking Water Contaminants

This introductory section summarizes the general approach used to evaluate the hazard of contaminants in drinking water. This text appears in all criteria documents to provide information relevant to the basic issue of how toxicological effects are quantified.

The quantification of toxicological effects of a chemical consists of separate assessments of noncarcinogenic and carcinogenic health effects. Chemicals which do not produce carcinogenic effects are believed to have a threshold dose below which no adverse, noncarcinogenic health effects occur, while carcinogens are assumed to act without a threshold.

Quantification of Noncarcinogenic Effects

In the quantification of noncarcinogenic effects, a Reference Dose (RfD), (formerly called the Acceptable Daily Intake (ADI)), is calculated. The RfD is an estimate of a daily exposure to the human population that is likely to be without appreciable risk of deleterious health effects, even if exposure occurs over a lifetime. The RfD is derived from a No-Observed-Adverse-Effect Level (NOAEL), or Lowest-Observed-Adverse-Effect Level (LOAEL), identified from a subchronic or chronic study, and divided by an uncertainty factor(s). The RfD is calculated as follows:

$$\text{RfD} = \frac{\text{(NOAEL or LOAEL)}}{\text{Uncertainty Factor(s)}} = \underline{\quad\quad} \text{ mg/kg bw/day}$$

Selection of the uncertainty factor to be employed in the calculation of the RfD is based on professional judgment, while considering the entire data base of toxicological effects for the chemical. In order to ensure that uncertainty factors are selected and applied in a consistent manner, the Office of Water (OW) employs a modification to the guidelines proposed by the National Academy of Sciences (NAS 1977, 1980a) as follows:

- An uncertainty factor of 10 is generally used when good chronic, or subchronic human exposure data identifying a NOAEL are available and are supported by good chronic, or subchronic toxicity data in other species.

- An uncertainty factor of 100 is generally used when good chronic toxicity data identifying a NOAEL are available for one or more animal species (and human data are not available), or when good chronic, or subchronic toxicity data identifying a LOAEL in humans are available.

- An uncertainty factor of 1,000 is generally used when limited or incomplete chronic, or subchronic toxicity data are available, or when good chronic, or subchronic toxicity data identify a LOAEL, but not a NOAEL for one or more animal species are available.

The uncertainty factor used for a specific risk assessment is based principally on scientific judgment rather than scientific fact and accounts for possible intra- and interspecies differences. Additional considerations not incorporated in the NAS/OW guidelines for selection of an uncertainty factor include the use of a less than lifetime study for deriving a RfD, the significance of the adverse health effect and the counterbalancing of beneficial effects.

From the RfD, a Drinking Water Equivalent (DWEL) can be calculated. The DWEL represents a medium specific (i.e., drinking water) lifetime exposure at which adverse, noncarcinogenic health effects are not anticipated to occur. The DWEL assumes 100% exposure from drinking water. The DWEL provides the noncarcinogenic health effects basis for establishing a drinking water standard. For ingestion data, the DWEL is derived as follows:

$$\text{DWEL} = \frac{(\text{RfD}) \times (\text{Body Weight in kg})}{\text{Drinking Water Volume in L/day}} = \underline{\quad}\ \text{mg/L}$$

where:

Body weight = assumed to be 70 kg for adult

Drinking water volume = assumed to be 2 liters per day for an adult

In addition to the RfD and the DWEL, Health Advisories (HAs) for exposures of shorter duration (One-day, Ten-day and Longer-term) are determined. The HA values are used as informal guidance to municipalities and other organizations when emergency spills or contamination situations occur. The HAs are calculated using a similar equation to the RfD and DWEL; however, the NOAELs or LOAELs are identified from acute or subchronic studies. The HAs are derived as follows:

$$\text{HA} = \frac{(\text{NOAEL or LOAEL}) \times (\text{bw})}{(\text{UF}) \times (\underline{\quad}\ \text{L/day})} = \underline{\quad}\ \text{mg/L}$$

Using the above equation, the following drinking water HAs are developed for noncarcinogenic effects:

1. One-day HA for a 10-kg child ingesting 1 L water per day.
2. Ten-day HA for a 10-kg child ingesting 1 L water per day.
3. Longer-term HA for a 10-kg child ingesting 1 L water per day.
4. Longer-term HA for a 70-kg adult ingesting 2 L water per day.

The One-day HA calculated for a 10-kg child assumes a single acute exposure to the chemical and is generally derived from a study of less than 7 days duration. The Ten-day HA assumes a limited exposure period of 1-2 weeks and is generally derived from a study of less than 30-days duration. A Longer-term HA is derived for both the 10-kg child and a 70-kg adult and assumes an exposure period of approximately 7 years (or 10% of an individual's lifetime). A Longer-term HA is generally derived from a study of subchronic duration (exposure for 10% of animal's lifetime).

Quantification of Carcinogenic Effects

The EPA categorizes the carcinogenic potential of a chemical, based on the overall weight-of-evidence, according to the following scheme:

- Group A: Human Carcinogen. Sufficient evidence exists from epidemiology studies to support a causal association between exposure to the chemical and human cancer.

- Group B: Probable Human Carcinogen. Sufficient evidence of carcinogenicity in animals with limited (Group B1) or inadequate (Group B2) evidence in humans.

- Group C: Possible Human Carcinogen. Limited evidence of carcinogenicity in animals in the absence of human data.

- Group D: Not classified as to Human Carcinogenicity. Inadequate human and animal evidence of carcinogenicity or for which no data are available.

- Group E: Evidence of Noncarcinogenicity for Humans. No evidence of carcinogenicity in at least two adequate animal tests in different species or in both adequate epidemiologic and animal studies.

If toxicological evidence leads to the classification of the contaminant as a known, probable or possible human carcinogen, mathematical models are used to calculate the estimated excess cancer risk associated with the ingestion of the contaminant in drinking water.

Quantification of Cancer Risk for Chemicals

For chemicals, the data used in these estimates usually come from lifetime exposure studies in animals. In order to predict the risk for humans from animal data, animal doses must be converted to equivalent human doses. This conversion includes correction for noncontinuous exposure, less than lifetime studies and for differences in size. The factor that compensates for the size difference is the cube root of the ratio of the animal and human body weights. It is assumed that the average adult human body weight is 70 kg and that the average water consumption of an adult human is 2 liters of water per day.

For contaminants with a carcinogenic potential, chemical levels are correlated with a carcinogenic risk estimate by employing a cancer potency (unit risk) value together with the assumption for lifetime exposure via ingestion of water. The cancer unit risk for chemicals is usually derived from a linearized multistage model with a 95% upper confidence limit providing a low dose estimate; that is, the true risk to humans, while not identifiable, is not likely to exceed the upper limit estimate and, in fact, may be lower. Excess cancer risk estimates may also be calculated using other models such as the one-hit, Weibull, logit and probit. There is little basis in the current understanding of the biological mechanisms involved in chemically-caused cancer to suggest that any one of these models is able to predict risk more accurately than any others. Because each model is based upon differing assumptions, the estimates which were derived for each model can differ by several orders of magnitude.

The scientific data base used to calculate and support the setting of cancer risk rate levels for chemicals has an inherent uncertainty due to the systematic and random errors in scientific measurement. In most cases, only studies using experimental animals have been performed. Thus, there is uncertainty when the data are extrapolated to humans. When developing cancer risk rate levels, several other areas of uncertainty exist, such as the incomplete knowledge concerning the health effects of contaminants in drinking water, the impact of the experimental animal's age, sex and species, the nature of the target organ system(s) examined and the actual rate of exposure of the internal targets in experimental animals or humans. Dose response data usually are available only for high levels of exposure, not for the lower levels of exposure closer to where a standard may be set. When there is exposure to more than one contaminant, additional uncertainty results from a lack of information about possible synergistic or antagonistic effects.

Quantification of Cancer Risk for Radionuclides

For radionuclides, human epidemiologic data rather than animal experiments form the basis of the cancer risk rate levels, which are derived as best estimates rather than as 95% upper confidence limits of the unit risk from a linearized multistage model. The true risks to humans may be higher or lower than the predicted risks, but the overall uncertainty is probably less than an order of magnitude. Because human data are used, the individual sites of cancer are predicted, as well as the total risk. Therefore, projections can be made both of cancer incidence and of cancer fatality, which are related for a given organ to site-specific survival data, which ranges from 90% survival (10% mortality) for thyroid cancer to 0% survival (100% mortality) for liver cancer (USEPA 1989b).

A. Noncarcinogenic Effects

There is little evidence for noncarcinogenic effects of exposure to radon or its decay products in humans (NAS 1988, ATSDR 1989). Respiratory and hematological effects of inhalation exposure to radon and radon decay products occur in animals at levels far above those causing lung cancer (ATSDR 1989). Therefore, USEPA considers that it is inappropriate to derive a Reference Dose for radon in drinking water.

B. Carcinogenic Effects

1. Categorization of Carcinogenic Potential

Radon is classified as a Group A carcinogen, a known human carcinogen. This classification is based on the known human carcinogenicity of ionizing radiation, supported by sufficient evidence from epidemiologic and experimental studies establishing the cancer-causing potential of exposure to radon and radon decay products in air.

2. Quantification of Carcinogenic Risk

Radon in potable water supplies results in exposure through ingestion due to drinking water from the tap and through inhalation due to release of radon into the air from household uses of water. The inhalation risk has generally been considered to dominate, but until recently, available data on the toxicokinetics of ingested radon were insufficient to estimate organ doses with any certainty (Cross et al. 1985).

Risks from Ingestion Exposure to Radon

No human or animal data are available to quantify the risk of ingestion of radon. However, external or internal irradiation is well-established as a cause of cancer, and quantitative estimates are available of the cancer risk associated with a given radiation dose for individual human organs (USEPA 1989b). Therefore, the approach to assessing the risk of ingested radon is to determine the individual organ doses of radiation based on radon toxicokinetics and then to apply the organ-specific risk factors to evaluate lifetime cancer risk for fatal and total cancers.

The toxicokinetics of ingested radon are discussed in Section III.A. Based on measurements of xenon kinetics performed by Correia et al. (1987), Crawford-Brown (1990) developed a model for the movement of radon through the human body. Ingested radon is transferred from the stomach to the small intestine, from which it is quickly absorbed into the bloodstream. Radon in blood circulates to all organs, and is quickly exhaled when it reaches the lung. Crawford-Brown (1990) calculated transfer rate constants for his 10-compartment model from simultaneous fitting of the data from Correia et al. (1987) for organ concentrations as a function of time and organ washout half-times. The model is shown in Figure III-1 and the fitted rate constants are listed in Table III-4.

Crawford-Brown (1990) used the model to calculate radon concentrations in organs as a function of time after radon ingestion. The concentration of radon in the intestinal wall (stomach, small intestine, ascending colon and descending colon) was calculated by assuming that there is a linear gradient between stomach concentration and capillary concentration and that the sensitive cells lie at a depth of 30 μm in a 40 μm layer. The concentration of radon in lung tissue was assumed to be equal to liver tissue, due to lung perfusion by portal blood.

The calculations of organ doses corresponding to calculated concentrations of radon are discussed in Section VII.B.1. The organ doses corresponding to an intake of 1 pCi of radon are given in Table VIII-1. These

Table VIII-1 Organ Doses of Alpha Irradiation from Ingestion of 1 pCi of Radon

Organ	Dose (rad/pCi)
Stomach	1.0E-08
Small Intestine	5.2E-09
Ascending Colon	7.4E-09
Descending Colon	4.1E-09
Liver	1.5E-09
Lung Alveoli	1.8E-09
General Tissue	6.7E-10

Adapted from Crawford-Brown 1990, with stomach dose multiplied by 0.60 to reflect removal of decay products beyond Po-218 prior to decay (Hursh et al. 1965).

values are based on the assumption that decay products decay at the site they are produced (Crawford-Brown 1990), except in the stomach (Hursh et al. 1965). The dose to the lung includes decay of radon in alveolar sacs prior to exhalation (Crawford-Brown 1990).

To estimate the risk from radon in drinking water using the results in Tables VIII-1, the total intake in pCi corresponding to a concentration of 1 pCi/L in drinking water must be calculated. The daily intake of radon depends upon the amount of radon remaining in water after handling in the preparation of beverages. As discussed in Section IV.C.1., radon release is relatively slow for cold, unagitated water, and quite rapid for hot or stirred water. Values ranging from 0 to 50% have been published for loss of radon from tap water during the process of filling a glass and drinking from it (von Dobeln and Lindell 1964, Hursh et al. 1965, Suomela and Kahlos 1972). For the purposes of assessing risk from radon consumption, it will be assumed that radon-containing water consists only of direct tap water, and that all tap-water based beverages have no residual radon due to heating, stirring and/or storage. It will also be assumed that on average, 20% of radon is lost during the processes of filling a glass from the tap and agitation while drinking (USEPA 1991b).

Available studies of tap water intake indicate that the average intake of direct tap water is about 0.6 to 0.7 L/day, and that about half of tap water is consumed as direct tap water (Pennington 1983, USEPA 1984, Ershow and Cantor 1989). This suggests that the best estimates of tap water consumption are about 0.7 L/day for direct tap water and 1.2 L/day for total tap water. For consistency with the practice of using 2 L/day for adult tap water consumption in the regulation of other drinking water contaminants, a value of

1 L/day for direct tap water is a reasonable, protective value, expressed to one significant figure (USEPA 1991b).

Using the above parameters, the lifetime intake of radon (I_{Rn}) from a drinking water source that contains 1 pCi/L is:

$$\begin{aligned} I_{Rn} &= 1\ \text{pCi/L} \times 1\ \text{L/day} \times 0.8 \times 365.25\ \text{d/y} \times 70\ \text{yr} \qquad \text{(VIII-1)} \\ &= 20{,}500\ \text{pCi} \end{aligned}$$

Thus, lifetime risk corresponding to a concentration of 1 pCi/L in drinking water is evaluated using a lifetime intake of 20,500 pCi. Table VIII-2 presents organ doses of alpha irradiation in rad per pCi/L, obtained by multiplying the values in Table VIII-1 by 20,500 pCi.

USEPA has calculated organ-specific lifetime risks of cancer incidence and fatality as a function of radiation dose (USEPA 1989b). Age- and sex-specific risk coefficients were derived for various organs, based on quantitative evaluation of epidemiological data on human cancer risk following exposure to several types of ionizing radiations (USEPA 1989b). Organ-specific risk coefficients were derived from values derived by the BEIR III committee of the National Academy of Sciences (NAS 1980b). The specific values used were mortality risk predicted by the linear-linear model with a 10-year minimum latency and a 30-year follow-up. Life-table methods based on U.S. mortality during 1969-1971 mortality data and a birth male:female ratio of 1.0511 were used, constrained by total risk at all sites (USEPA 1989b). This procedure was used to derive relative risk coefficients for fatal cancers except for leukemia and bone cancer, for which absolute risks with a 2-year minimum induction period and a 25-year expression period were calculated from

Table VIII-2 Lifetime Organ Doses of Alpha Irradiation per pCi/L of Radon in Drinking Water

Organ	Dose (rad/pCi)
Stomach	2.1E-04
Small Intestine	1.1E-04
Ascending Colon	1.5E-04
Descending Colon	8.3E-05
Liver	3.0E-05
Lung Alveoli	3.7E-05
General Tissue	1.4E-05

Calculated from entries in Table VIII-1 multiplied by 20,500 pCi per pCi/L in drinking water (see text).

the BEIR III absolute risk coefficients (NAS 1980b). The leukemia and bone cancer risk coefficients were combined using a relative biological effectiveness of 8 for alpha particles. Mortality to incidence ratios were derived from values provided by BEIR III (USEPA 1989b).

Risks for high-LET radiation are derived from risks for low-LET radiation by multiplying by a relative biological effectiveness of 8 (USEPA 1989b). This is the appropriate value for comparing high-LET risks to the low-LET risks derived by USEPA (1989b). ICRP recommends a value of 20 when multiplying by ICRP-derived low-LET risks (ICRP 1977). The ICRP-derived low-LET risks incorporate a 2.5-fold reduction for low doses and dose rates (ICRP 1977). However, the USEPA (1989b) low-LET risk numbers do not incorporate this 2.5-fold reduction, because human data on breast and thyroid cancer do not support such a reduction and because there is an essentially equal reduction in predicted low-LET risk due to the use of a linear rather than a linear-quadratic risk projection model (USEPA 1989b). Human data on alpha emitters also support a linear risk projection model (except for bone sarcomas among radium-exposed watch dial painters). The appropriate relative biological effectiveness to use with USEPA (1989b) risk coefficients for low-LET radiation is 20/2.5 = 8.

Averaged risk coefficients for high-LET radiation are listed in Table VIII-3 in units of risk per rad for fatal and total cancers for the organs in the model of Crawford-Brown (1990). These risk per rad values are multiplied by the organ-specific rad per pCi/L values in Table VIII-2 to provide estimates of lifetime risk per pCi/L in drinking water. The

Table VIII-3 Organ-Specific Lifetime Cancer Risks
from Alpha Irradiation

Organ	Risk/rad Fatality	Risk/rad Incidence
Stomach	3.7E-4	4.8E-4
Small Intestine	3.7E-5	6.9E-5
Ascending Colon	7.3E-5	1.4E-4
Descending Colon	7.3E-5	1.4E-4
Liver	4.0E-4	4.0E-4
Pulmonary Lung	5.7E-4	6.0E-4
All other tissue	1.6E-3	3.2E-3

Adapted from USEPA (1989b) Tables 6-6 and 6-7. Intestinal risk apportioned among small intestine, ascending colon and descending colon using weights of 0.2, 0.4 and 0.4 respectively (USEPA 1989b, Table 5-4).

results in risks per pCi/L of radon in water, are presented in Table VIII-4. Total risks are estimated to be 1.5E-7 per pCi/L for fatal cancers and 2.2E-7 for total cancers.

Risks from Inhalation Exposure to Radon Decay Products

The relationship between exposure to radon decay products and increased risk of lung cancer has been demonstrated in many studies of underground miners (see Section VI.B.2). These data have been analyzed quantitatively by several investigators, both for individual cohorts and for several cohorts combined. The Agency's risk estimate is based upon two recent authoritative analyses, those of the BEIR IV committee of the National Academy of Sciences (NAS 1988) and of the ICRP 50 committee of the International Commission on Radiological Protection (ICRP 1987). Both of these are relative risk models that assume a multiplicative interaction between radon exposure and smoking in causing lung cancer.

ICRP 50 Model. The ICRP 50 model is based on a combined analysis of Colorado Plateau, Ontario and Czech mining cohorts (see Section VI.B.2). The ICRP 50 model is expressed as:

$$r(a) = r_0(a)\ (1 + B(a)W) \qquad \text{(VIII-2)}$$

where:

$r(a)$ = lung cancer mortality rate at age a due to all causes

$r_0(a)$ = lung cancer mortality rate in the absence of radon exposure

Table VIII-4 Organ-Specific Lifetime Cancer Risks
from Ingestion of Radon in Drinking Water

Organ	Risk/pCi/L	
	Fatality	Incidence
Stomach	7.7E-8	1.0E-7
Small Intestine	3.9E-9	7.3E-9
Ascending Colon	1.1E-8	2.1E-8
Descending Colon	6.1E-9	1.1E-8
Liver	1.2E-8	1.2E-8
Pulmonary Lung	2.1E-8	2.2E-8
All other tissue	2.2E-8	4.3E-8
Total	1.5E-7	2.2E-7

Calculated from entries in Tables VIII-2 and VIII-3.

$B(a)$ = age-dependent relative risk coefficient in %/WLM

W = cumulative radon exposure in WLM up to 10 years before age a

The ICRP 50 committee recommended a value of B of 1.9 %/WLM for ages between 0 and 19 and a value of 0.64 %/WLM for ages 20 and above. These recommended coefficients assume a 20% lower risk in homes than in mines for equal WLM, based on dosimetric considerations. The three-times-higher risk for children is based on greater sensitivity at younger ages to lung cancers induced by external radiation exposure among atomic bomb survivors, together with consideration of the greater radiation dose to the lung among children. The ICRP 50 committee calculated the proportion of observed lung cancers due to household radon exposure, assuming an average population exposure of 0.19 WLM/year, and subtracted these cancers from the baseline rate. Thus, the ICRP 50 model is a relative risk model adjusted for background exposure and for a 20% dosimetry difference in mines and homes.

BEIR IV Model. The BEIR IV model is based on a combined analysis of the Colorado Plateau, Ontario, Saskatchewan and Malmberget cohorts (see Section VI.B.2). The BEIR IV model is expressed as:

$$r(a) = r_0(a)\ [1 + B(a)(W_1 + 0.5W_2)] \qquad \text{(VIII-3)}$$

where:

$r(a)$ = lung cancer mortality rate at age a due to all causes

$r_0(a)$ = lung cancer mortality rate in the absence of radon exposure

$B(a)$ = age-dependent relative risk coefficient in %/WLM

W_1 = cumulative radon exposure in WLM between 5 and 15 years before age a

W_2 = cumulative radon exposure in WLM more than 15 years before age a

The BEIR IV committee recommended values of B of 3%/WLM for ages below 55 years, 2.5%/WLM for ages between 55 and 64 years and 1%/WLM for ages above 65 years. These coefficients assume that risks in homes and mines are equal for equal WLM, based on dosimetric considerations. No adjustment was made to baseline risks for household radon exposure, but it was noted that this correction would be small (NAS 1988). Thus, the BEIR IV model is a relative risk model that is not adjusted for background exposure and that assumes no dosimetry difference between mines and homes.

USEPA Estimate. Based on advice from the Radiation Advisory Committee of the EPA Science Advisory Board, the Agency's current estimate for radon risk is based on combining the ICRP 50 and BEIR IV models to provide a central estimate of household radon exposure (USEPA 1989b). Before the two models can be combined, the adjustments for background exposure and dosimetry in mines and homes must be made consistent. The Agency assumes that baseline lung cancer rates should be adjusted for background radon exposure, as in the ICRP 50 model, and that risks per WLM should be considered equal in mines and in homes, based on the analysis by the BEIR IV committee (NAS 1988). Thus, the dosimetry must be adjusted in the ICRP 50 model and the background lung cancer rates must be adjusted in the BEIR IV model.

Adjusting the dosimetry in the ICRP 50 model involves using values of B of 2.4 %/WLM for children and 0.8 %/WLM for adults in equation VIII-2. Using these values in a lifetable calculation using 1980 U.S. vital statistics, the ICRP 50 model predicts lifetime excess lung cancer mortality of 955 deaths per million person-WLM for children and 420 deaths per million person-WLM for adults (USEPA 1989b). The background adjustment to the BEIR IV model assumes an average background exposure rate of 0.25 WLM/year (Puskin and Yang 1988). The correction is model-specific, and involves subtracting from the observed age-specific lung cancer mortality rate the number of radon-induced lung cancers calculated for a cumulative exposure of 0.25 WLM/year using the age-specific risk coefficient. Using the adjusted BEIR IV model in a lifetable calculation using 1980 U.S. vital statistics, predicted lifetime excess lung cancer mortality is 334 deaths per million person-WLM for children and 305 deaths per million person-WLM for adults (USEPA 1989b).

As a central estimate of risk from indoor radon exposure, USEPA averages the adult lifetime excess lung cancer mortality from the adjusted ICRP 50 and BEIR IV models, 420 and 305 deaths per million person-WLM, yielding a value of 360 deaths per million person-WLM of exposure, with an estimated range of 140 720 deaths per million person-WLM (USEPA 1989b). This risk estimate is currently undergoing review by the Science Advisory Board.

In estimating indoor exposures to radon decay products, an equilibrium factor of 0.5 and an occupancy fraction of 75% are assumed (see

Section III.B.2). With these assumptions, exposure to 1 pCi/L of radon in air corresponds to an exposure of 0.19 WLM/year, and to 13 WLM in a 70-year lifetime. As discussed in Section IV.D, for assessing risks from radon in drinking water, an important parameter is the increase in indoor radon concentration due to radon in the water supply. This parameter is termed the transfer factor. The Agency uses an average transfer factor of 10,000:1 to evaluate inhalation exposure (USEPA 1991b). With this transfer factor, 1 pCi/L of radon in water gives rise to 1E-4 pCi/L of radon in air, corresponding to a lifetime exposure of 1.3E-3 WLM per pCi/L of radon in water. Multiplying by the risk coefficient of 360 deaths per million person-WLM yields a risk of 4.8E-7 deaths per pCi/L in water. Adjusting for 6% survival of lung cancer (USEPA 1989b) yields a total cancer risk of 5.1E-7 per pCi/L in water.

Smokers are at substantially higher risk than nonsmokers for developing lung cancer from inhaled radon decay products (see Section VI.C). Using the risk multipliers presented in Table VI-1 (USEPA 1990b), the risk per pCi/L from inhalation of radon decay products released from household uses of water is 9.6E-8 (fatal) and 1.0E-7 (total) for nonsmokers, 3.8E-7 (fatal) and 4.1E-7 (total) for former smokers and 8.6E-7 (fatal) and 9.2E-7 (total) for current smokers.

Risks from Inhalation Exposure to Radon Gas

Risks from inhalation of radon gas are assessed using the RADRISK model developed by the USEPA Office of Radiation Programs (ORP) (Sullivan et al. 1981, USEPA 1989b). An outline of the methodology is provided in Sections III.B.1 (biokinetic model) and VII.B.1 (dosimetric model). Important features are the use of a metabolic compartmental model to calculate absorption, distribution, metabolism and excretion of radon and radon decay products, the separate calculation of absorbed dose rate for low-LET and high-LET radiation, the use of the organ-specific risk coefficients derived by the BEIR III committee (NAS 1980b) based on a synthesis of information on radiogenic cancers, and the use of life-table methods to convert age- and sex-specific yearly risks to total lifetime risk.

The dose rates calculated by the RADRISK model for various organs are listed in Table VIII-5, expressed as the annual dose rates in year 70 resulting from a lifetime constant inhalation of 1 pCi/year of radon. The calculated dose rate in a given year for continuous intake is numerically equal to the integrated lifetime dose for the cumulative intake until that year (ICRP 1971). The RADRISK model actually calculates the annual organ-specific dose rates at various times during the life span, not just the dose in year 70, for purposes of assessing lifetime risk (Sullivan et al. 1981, Dunning et al. 1980, USEPA 1989b).

USEPA has derived age- and sex-specific risk coefficients synthesizing the results of many epidemiologic studies on radiation-induced cancer risks in various organs, as described in the section concerning risk from radon ingestion. Averaged risk coefficients used in the calculations for radon gas inhalation in the RADRISK model are listed in Table VIII-6 for high-LET and low-LET radiation in units of risk per rad for fatal and total cancers. The RADRISK model uses age- and sex-specific organ dose rates and risk coefficients together with life-table methods to calculate lifetime risk by

Table VIII-5 Annual Organ Doses in the 70th Year Predicted by the RADRISK Model from Inhalation of 1 pCi/year of Radon

Organ	LET	Dose (rad/yr)/(pCi/yr)
Pulmonary lung	Low	2.04E-11
	High	3.33E-10
Stomach	Low	1.14E-11
	High	1.43E-10
Intestine	Low	1.47E-11
	High	1.43E-10
Kidneys	Low	5.95E-12
	High	4.15E-11
Liver	Low	5.43E-12
	High	3.97E-11
Breast	Low	1.09E-11
	High	1.43E-10
Pancreas	Low	1.21E-11
	High	1.43E-10
Red marrow	Low	6.11E-12
	High	4.27E-11
Bone surface	Low	4.79E-12
	High	2.06E-10
Thyroid	Low	1.10E-11
	High	1.43E-10
All other tissue	Low	1.21E-11
	High	1.43E-10

Table VIII-6 Organ-Specific Lifetime Cancer Risks Used in the RADRISK Model from High-LET and Low-LET Irradiation

		Risk/rad	
Organ	LET	Fatality	Incidence
Pulmonary lung	Low	7.0E-5	7.5E-5
	High	5.7E-4	6.0E-4
Stomach wall	Low	4.6E-5	6.0E-5
	High	3.7E-4	4.8E-4
Intestine	Low	2.3E-5	4.3E-5
	High	1.8E-4	3.4E-4
Kidneys	Low	1.8E-5	4.3E-5
	High	1.4E-4	3.4E-4
Liver	Low	5.0E-5	5.0E-5
	High	4.0E-4	4.0E-4
Breast	Low	5.5E-5	1.4E-4
	High	4.4E-4	1.1E-3
Pancreas	Low	3.5E-5	3.8E-5
	High	2.8E-4	3.1E-4
Red marrow	Low	4.5E-5	4.5E-5
	High	3.6E-4	3.6E-4
Bone surface	Low	2.5E-6	2.5E-6
	High	2.0E-5	2.0E-5
Thyroid	Low	6.4E-6	6.4E-5
	High	5.1E-5	5.1E-4
Esophagus	Low	9.1E-6	9.1E-6
	High	7.3E-5	7.3E-5
Lymphoma	Low	1.4E-5	1.9E-5
	High	1.1E-4	1.5E-4
All other tissue	Low	1.9E-5	3.4E-5
	High	1.6E-5	2.7E-4

Adapted from USEPA (1989b) Tables 6-6 and 6-7.

organ resulting from high-LET and low-LET radiation. The excess lifetime risks predicted by the RADRISK model for inhalation of 1 pCi of radon gas are 7.2E-13 for total cancers and 4.7E-13 for fatal cancers (USEPA 1989b).

As stated in the previous discussion of risks from inhaled radon decay products, the Agency uses an average transfer factor of 10,000:1 to evaluate inhalation exposure to radon released from household uses of water (USEPA 1991b). With this transfer factor, 1 pCi/L of radon in water gives rise to 1E-4 pCi/L of radon in air. Assuming 75% occupancy (see Section III.B.2) and a breathing rate of 22,000 L/day (USEPA 1989b), an individual inhales a total of 4.2E+8 L in a 70-year lifetime. Thus, the amount of radon gas inhaled over a 70-year lifetime from 1 pCi/L of radon in water is 4.2E+4 pCi. Using the risk factors of 7.2E-13 for total cancers and 4.7E-13 for fatal cancers per pCi of radon gas inhaled (USEPA 1989b), the risks per pCi/L in water are 3.0E-8 for total cancers and 2.0E-8 for fatal cancers.

Total Cancer Risk

Adding together fatal cancer risks for ingestion of radon (1.5E-7 per pCi/L), inhalation of radon decay products (4.9E-7 per pCi/L) and inhalation of radon gas (2.0E-8 per pCi/L) yields a fatal cancer risk of 6.6E-7 per pCi/L. Similarly, adding total cancer risk for ingestion of radon (2.2E-7 per pCi/L), inhalation of radon decay products (5.1E-7 per pCi/L) and inhalation of radon gas (3.0E-8 per pCi/L) yields a total cancer risk of 7.6E-7 per pCi/L.

C. Genetic and Developmental Effects

Exposure to ionizing radiations can cause mutations in germ cells (oocytes and spermatogonia) leading to hereditary defects, and can damage embryos and fetuses exposed in utero leading to pre-implantation loss, mental retardation or malformations (USEPA 1989b). These effects are primarily established in experimental animals exposed to gamma irradiation, but a risk of mental retardation and microcephaly has been established among Japanese A-bomb survivors exposed in utero (USEPA 1989b). No direct evidence exists that exposure to radon causes hereditary genetic defects or teratogenesis (see Sections V.C and VI.B.1). However, estimates of genetic risks can be made by extrapolation of observed effects of low-LET irradiation. The Agency has reviewed several estimates of the risk factors for low-LET and high-LET irradiation and has developed the risk factors shown in Table VIII-7 (USEPA 1989b). These risk factors are based primarily on evaluations by the National Academy of Sciences (NAS 1980b, 1988). Risk factors were not developed for teratogenic or somatic risk for high-LET irradiation because of the lack of a plausible dosimetry for the developing fetus.

Since no data are available to estimate the teratogenic or somatic risk from high-LET irradiation, the assessment of risk from radon exposure is limited to genetic defects. Risk factors for genetic defects are expressed relative to the 30-year cumulative dose to ovaries and testes. For ingestion of radon in water, the model of Crawford-Brown (1990) did not include gonadal tissue as a separate compartment. It is assumed that these doses are similar to the alpha particle doses calculated for general body tissue,

Table VIII-7 Radiation Risk Factors for Genetic and Developmental Effects

Endpoint	Significant Exposure Period	Risk per rad	
		Best Estimate	Range
Low LET			
Teratogenic[a]			
Severe mental retardation	Weeks 8 to 15 of gestation	4.0E-3	2.5E-3 - 5.5E-3
Somatic			
Fatal cancers	Weeks 0 to 40 of gestation	6.0E-4	1.8E-4 - 1.8E-3
Genetic			
Severe hereditary defects, all generations	30-year reproductive generation	2.6E-4	6.0E-5 - 1.1E-3
High LET			
Genetic			
Severe hereditary defects, all generations	30-year reproductive generation	6.9E-4	1.6E-4 - 2.9E-3

Adapted from USEPA (1989b) Table 6-27.

[a] The range assumes a linear, non-threshold dose response. However, it is plausible that a threshold may exist for teratogenic effects.

6.7E-10 rad/pCi (see Table VIII-1). Over a 30-year period, a concentration of 1 pCi/L of radon in water leads to a total radon ingestion I_{Rn} of:

$$I_{Rn} = 1\ \text{pCi/L} \times 1\ \text{L/day} \times 0.8 \times 365.25\ \text{d/y} \times 30\ \text{yr} \qquad \text{(VIII-4)}$$
$$= 8{,}800\ \text{pCi}$$

Thus, the gonadal dose of alpha radiation from ingestion is equal to 5.9E-6 rad per pCi/L of radon in water.

The RADRISK model calculates dose to gonadal tissue from inhalation of radon gas (USEPA 1989b). The annual doses are 1.20E-11 rad/year per pCi/year for low-LET radiation and 1.43E-10 rad/year per pCi/year for high-LET radiation, which are virtually identical to the doses for general tissue (see Table VIII-5). Assuming a transfer factor of 10,000:1, a breathing rate of 22,000 L/day and a occupancy fraction of 75%, the 30-year cumulative radon gas inhalation is 1.8E4 pCi, and so the 30-year cumulative doses to gonadal tissue are 2.2E-7 (low-LET) and 2.6E-6 (high-LET) rad per pCi/L of radon in water.

Table VIII-8 summarizes the predicted gonadal doses. Note that the dose from inhaled radon decay products is not included, because models have not been developed to predict irradiation of organs other than the lung from inhaled radon decay products. Note also that an estimate of dose from low-LET radiation is available only for inhalation of radon gas, not ingestion of radon.

Table VIII-8 30-Year Cumulative Doses to Germ Cells Predicted for Ingestion and Inhalation of Radon Gas

Route	LET	Gonadal dose (rad)/(pCi/L in water)	Source
Ingestion	Low	Not available	Crawford-Brown 1991
	High	5.9E-6	
Inhalation	Low	2.2E-7	USEPA 1989b
	High	2.6E-6	
Total	Low	2.2E-7	
	High	8.5E-6	

Germ cell mutation risks calculated from the estimated doses (Table VIII-8) and risk factors (Table VIII-7) are shown in Table VIII-9. These risks are about 0.8% of the predicted cancer risks for ingestion of radon, inhalation of radon gas and inhalation of radon decay products. Uncertainty in the estimates of genetic and developmental effects are discussed in Chapter IX.

D. Summary

Table VIII-10 summarizes the estimated excess total cancer risk calculated to correspond to various levels of radon in water.

Table VIII-9 Germ Cell Mutation Risks from Lifetime Exposure to 1 pCi/L Radon in Drinking Water

Parameter	LET	Value
Dose in rad/(pCi/L)[a]	Low	2.2E-7
	High	8.5E-6
Risk per pCi/L[b]	Low	5.7E-11
	High	5.9E-9
	Total	5.9E-9

[a] From Table VIII-8

[b] Calculated from the previous entries multiplied by the coefficients in Table VIII-7.

Table VIII-10 Summary of Quantification of Toxicological Effects for Radon in Drinking Water

Value	Drinking water concentration of radon (pCi/L)	Reference
Excess cancer risk		
Total Cancers		
10^{-4}	130	USEPA (1989b)
10^{-5}	13	USEPA (1989b)
10^{-6}	1.3	USEPA (1989b)
Fatal Cancers		
10^{-4}	150	USEPA (1989b)
10^{-5}	15	USEPA (1989b)
10^{-6}	1.5	USEPA (1989b)

IX. UNCERTAINTY ANALYSIS

The health effects following exposure to radon and ionizing radiations in general are very well established from both human and animal data. Most of the uncertainty arises in evaluating the doses to target organs received from environmental exposures. The remainder of this section briefly describes the assumptions and parameters contributing uncertainty to the quantification of the toxicological effects of radon in drinking water.

A. Range of Assumptions and Models

1. Noncancer Effects

It is assumed that noncancer health effects would not be of concern at levels of exposure from radon in household water. Respiratory and hematological effects of inhalation exposure to radon and radon decay products occur in animals at levels far above those causing lung cancer (ATSDR 1989). Noncancer health effects observed in miners exposed to radon and radon decay products by inhalation include chromosomal aberrations and possible changes in the birth weight offspring (NAS 1988). The chromosomal aberrations, which are likely to be related to the mutagenicity of ionizing radiation, do not appear to be a more sensitive indicator than lung cancer of the genotoxicity of radon and radon decay products, and so consideration of carcinogenicity should be sufficiently protective against chromosomal aberrations. The evidence for a change in the birth weight of offspring is not strong, but possible reproductive effects are analyzed based on germ cell mutations (Section VIII.C).

2. Cancer Risk from Radon Ingestion

The key assumptions made in evaluating the cancer risk of ingestion of radon from water supplies are the carcinogenicity of radon by the ingestion route, the shape of the dose-response curve, derivation of organ-specific doses per unit radon ingestion and the derivation of organ-specific risks per unit alpha dose. Uncertainties due to parameter variability are discussed in Section IX.B.1.

Ingestion Carcinogenicity of Radon and Decay Products

The carcinogenicity of ingested radon or radon decay products has not been established by direct evidence, but all radionuclides are classified as Group A carcinogens based on their property of emitting ionizing radiation (USEPA 1991e). Therefore, there is little uncertainty in assuming that ingested radon and decay products are carcinogenic.

Shape of the Dose-Response Curve

The assumption was made in assessing ingestion risk that the dose-response curve is linear. A linear response to alpha-particle exposure by ingestion is generally consistent with the human and animal data, with the exception of the dose-squared incidence of bone sarcomas following ingestion of radium (USEPA 1989b). Thus, although no direct data are available for ingested radon, the uncertainty contributed by possible nonlinearities in the dose-response curve is probably less than a factor of two.

Derivation of Dose per Unit Radon Ingestion

The Agency has evaluated ingestion risks by using estimates of organ-specific alpha doses derived from a toxicokinetic model developed by Crawford-Brown (1990), based on data for body distribution of xenon measured by Correia et al. (1987). Crawford-Brown (1990) evaluated the uncertainty in calculated dose per unit intake due to assumptions concerning calculation of radon concentration in organs per unit ingestion, location of sensitive cells in the gastrointestinal tract, recirculation of progeny and effect of age on dose. This analysis assumed that quantities are lognormally distributed and independent. Organ concentrations per unit ingestion were estimated to have a geometric standard deviation (GSD) of 1.5, based on the large sample size in the Correia et al. (1987) study. Uncertainty concerning the location of sensitive cells was analyzed based on a difference of a factor of 5 between radon concentrations in the gastrointestinal lumen and blood, and assigned a GSD of 2. The recirculation of progeny was calculated to increased doses by less than 10%, and no GSD was assigned. The uncertainty due to assumptions concerning changes in dose with age was estimated to have a GSD of 2. Crawford-Brown (1990) estimated that the composite GSD was 3.1 for mean dose per unit intake. Estimates of other investigators were within a factor of 3 of the estimates of Crawford-Brown (1990). Hess and Brown (1991) considered the individual variation in dose per unit intake, and found a 50-fold difference between the highest and lowest calculated whole-body dose per unit intake among 38 volunteers.

Crawford-Brown (1990) also considered the uncertainty contributed by not evaluating risks from ingestion of radon decay products present in drinking water. For comparison of risks, it was assumed that all decay products had an activity equal to radon, such as would be achieved by secular equilibrium between radon and its decay products. The highest risk from the short-lived decay products ((Po-218, Pb-214, Bi-214 and Po-214) is from Bi-210, and is a factor of 8 lower than the risk from Rn-222. However, the risk from the long-lived decay products (Pb-210, Bi-210, Po-210) is more significant, and is 200 times higher for Pb-210 than for Rn-222. The half-life of Pb-210 is relatively long (22.3 years), which implies that geologic separation would prevent most aquifers from being in secular equilibrium. If substantial amounts of the long-lived decay products are in a drinking-water supply, they would be identified by the gross alpha screen because of the alpha emission of Po-210. No data were located concerning the co-occurrence of radon and long-lived decay products in drinking water supplies, and so the potential magnitude of the uncertainty due to ingestion of long-lived decay products cannot be evaluated.

Organ Risk Model Formulation

The dose estimates derived by Crawford-Brown (1990) were combined with estimates of organ-specific risks for alpha irradiation derived from data on risks from both high-LET and low-LET irradiations (USEPA 1989b). The assumptions used in calculating organ risk per unit dose were developed by the Agency with extensive consideration of work by ICRP and NAS and of advice from the Radiation Advisory Committee of the EPA Science Advisory Board. Uncertainties in the model formulation are addressed in USEPA (1989b), Chapter 6. These uncertainties include the shape of the dose-response curve, the effect of dose rate for low-LET and high-LET irradiations, the risk projection models (use of relative risk for all cancers except use of absolute risk for leukemia and bone cancers), the use of lifetable methods to assess lifetime risk, the use of a single value for relative biological effectiveness of high-LET irradiations for all organs and the implications of the revised dosimetry for low-LET irradiations from the Japanese A-bomb survivor data. The magnitude of these uncertainties was not addressed, but it was concluded that the overall projections of the RADRISK model were not biased low in a serious way for internally-deposited alpha emitters (USEPA 1989b).

3. Cancer Risk from Inhalation of Radon and Radon Decay Products

The key assumptions made in evaluating the cancer risk resulting from release of radon from household water supplies are the carcinogenicity of radon and radon decay products by the inhalation route, the shape of the dose-response curve, the relationship between water and air radon concentrations and the choice of a model for evaluating inhalation risk.

Inhalation Carcinogenicity of Radon and Decay Products

There is very little uncertainty concerning the capability of ionizing radiation in general and radon decay products in particular to cause cancer in humans. This high degree of certainty is reflected in the classification of radon as a Group A carcinogen, a known human carcinogen.

Shape of the Dose-Response Curve

Inhalation risks were assumed to have a linear dose-response curve. Both epidemiologic and animal data are consistent with a linear response except at very high exposures. An approximately linear relationship has been observed in miner cohorts down to levels of cumulative exposure that correspond to an average of about one alpha track traversal per cell nucleus (Harley 1988). This observed linearity is consistent with the hypothesis that lung cancers induced by radon decay products may originate from a single alpha track event, which would imply linearity at lower doses and dose rates (USEPA 1990b). The magnitude of the effect of any uncertainty concerning the shape of the dose response curve is reduced by the fact that the lowest dose at which excess lung cancers have been observed in miner cohorts is only slightly higher than the exposures experienced by members of the general population (USEPA 1990b). Overall, the uncertainty contributed by possible nonlinearities in the dose-response curve is probably less than a factor of two.

Relationship Between Water and Air Radon Levels

A number of studies have established that the presence of radon in water supplies causes elevated radon levels in household air (McGregor and Gourgon 1980, Becker and Lachajczyk 1984, Hess et al. 1990). Quantitative relationships can best be measured when water dominates as the source of indoor radon. To extrapolate the relationship at lower water levels requires identifying controlling variables. Available analyses have suggested that the influence of radon in water on radon in household air can be quantified in terms of a transfer factor in units of $(\text{pCi}/L_a)/(\text{pCi}/L_w)$ (Becker and Lachajczyk 1984, Nazaroff et al. 1987). The transfer factor is assumed to be a function of the rate of household water use, the use-weighted average transfer efficiency of radon from water to air, the volume of air in a house and the rate of exchange between indoor and outdoor air (Becker and Lachajczyk 1984, Nazaroff et al. 1987).

Experimental data generally confirm that these are the most important variables, but uncertainty is introduced by the assumption that the relevant quantity to be calculated is the average radon concentration in household air. Transient high concentrations of radon can exist when water is used in an enclosed space (e.g., shower stall, laundry room). Based on models developed by McKone (1987) and Small et al. (1990), concentrations in shower stalls can exceed average concentrations in household air by as much as a factor of 1,000, when successive long showers are taken in a small, poorly-ventilated shower stall (McKone 1987). The actual exposure to elevated concentrations will depend upon how much time a resident spends in the relevant locations, which clearly would be quite variable over the population. As described in Section IV.C., an analysis of the underprediction by the transfer factor of exposures during showers indicated that the underprediction was typically about 50% (USEPA 1991b).

For exposures to radon decay products, additional uncertainty is introduced because of possible differences in the dosimetry of radon decay products in shower air compared to general household air (USEPA 1991b). Exposure conditions in a shower are likely to differ in parameters such as the equilibrium factor, unattached fraction, aerosol size distribution and aerosol deposition characteristics. If the equilibrium factor is the most important influence on lung dose, the dose in a shower could be considerably less than in the general home environment, particularly if the primary fates of decay products that were produced was to attach to large, non-respirable drops of water or to the walls of the shower. On the other hand, the dose in a shower could be equal to or even higher than that in general home environment, if most decay products remained as ultrafine aerosols or became attached to mist particles that deposited with high efficiency in the respiratory tract. It seems most likely that the actual dose to sensitive target cells would be lower in a shower than at a comparable concentration of radon gas in the general home environment, but further investigations of the dosimetry of radon decay products in the showering environment are needed to quantify differences.

Model Used to Quantify Inhalation Risk of Radon Decay Products

For evaluating risk from inhalation of radon decay products, the Agency estimates exposure based on typical house conditions and estimates risk using human epidemiologic studies of underground miners. Uncertainty in using this mathematical model can be divided into model formulation, discussed below, and parameter variability, discussed in Section IX.B.2.

Inhalation Risk Model Formulations

Inhalation risk of radon and radon decay products is evaluated by extrapolation from data on lung cancer incidence among cohorts of underground miners rather than by the use of a dosimetric approach, based on recommendations from the Radiation Advisory Committee of the EPA Science Advisory Board (USEPA 1989b). A dosimetric approach was considered less accurate because of uncertainty in evaluating dose to target cells in the lung per unit exposure. Such dosimetric models have been employed to evaluate the need to adjust for differences in exposure between mines and homes at a given radon concentration (NAS 1988, 1991). Some uncertainty is introduced by the assumption that the lung is the only organ for which cancer risk is evaluated, because epidemiologic studies of underground miners may not be sensitive enough to detect increases in other forms of cancer, such as leukemia (NAS 1988).

The Agency's model for evaluating inhalation risk of radon decay products is based on the assumption that the best estimate of lung cancer risk is obtained by averaging the models recommended two expert committees (the BEIR IV and ICRP 50 committees), adjusted to be similar in correcting for background radon exposure and for differences in dosimetry between mines and homes. This approach is being reviewed by the Science Advisory Board, concerning the use of the ICRP 50 model and the relative dosimetry in mines and homes.

Both the BEIR IV and ICRP 50 models are relative risk models; that is, they assume that radon exposure multiplies the underlying risk of lung cancer. Neither model attempted to analyze radon risk using biologically-based two-stage or multi-stage models that have been developed to described the age-dependence of spontaneous and smoking-induced lung cancer (Chu 1987). The Radiation Advisory Committee concluded that a relative risk model was better supported by the miner data than an absolute risk model. However, it should be noted that the BEIR IV model includes a 40% reduction in risk above age 65. This is the age at which the rates of spontaneous and smoking-induced lung cancers begin to rise steeply (NAS 1988), and so this factor brings the BEIR IV model closer to an absolute risk model. The greatest uncertainty in using a relative risk model concerns the effect of gender and smoking, because females have a considerably lower baseline risk of lung cancer than males, and nonsmokers have a considerably lower baseline risk of lung cancer than smokers. The BEIR IV committee indicated that the doubling dose for females would be about three times lower for an additive rather than a relative risk model. No quantitative analysis of the effect of assuming an additive interaction for cigarette smoking was done by the BEIR IV committee, but it was noted that predicted risks would drop somewhat for smokers and rise considerably for nonsmokers (NAS 1988).

The two models both assume that risk is influenced by age. The ICRP 50 model assumes that exposures up to age 19 are three times as potent as exposures thereafter. This assumption was based on consideration of the higher relative risk of lung cancer among atomic bomb survivors irradiated as children, and also upon the greater dose per unit exposure due to differences in child breathing rate. Recently, the BEIR V committee concluded that this increased risk may fall off sharply with time since exposure, but that the evidence was not conclusive (NAS 1990). The assumption of a three-times higher risk during childhood is a major factor contributing to the higher risks projected by the ICRP 50 model compared to the BEIR IV model, and if children are in fact not more sensitive, then risks would be lower than projected by up to 20%.

The BEIR IV committee analyzed the uncertainty contributed by modification of risk with both age at risk and time since exposure. The BEIR IV model has risks 20% higher below age 55 and 40% lower after age 65, compared to risks between ages 55 and 65. The model also assumes that risk is half as great for exposures more than 15 years in the past compared to exposures 5 to 15 years in the past. The BEIR IV committee fit a constant relative risk model to the miner cohort data for comparison to the preferred model, and concluded that predicted risks from lifetime exposure would be higher by about 50% for the constant relative risk model.

Model Used to Quantify Inhalation Risk of Radon Gas

Only one model was available for assessing the risk of inhalation of radon gas, the RADRISK model (USEPA 1989b). That model was developed by USEPA specifically for the purpose of assessing risk of lifetime exposure to radionuclides by members of the general population. Uncertainty in using a mathematical model can be divided into model formulation, discussed below, and parameter variability, discussed in Section IX.B.3.

Dosimetric Model Formulation

The RADRISK model calculates the dose to target organs from ingestion of radon using the ORNL model for noble gas metabolism (Bernard and Snyder 1975) and the ICRP models for alkaline-earth metabolism, with certain modifications (see Section III.B.1), together with calculations of absorbed dose, also based on ICRP models with minor modifications (see Section VII.B.1). USEPA (1989b) contains an extensive discussion of the uncertainty associated with the ICRP models (pp. 5-20 to 5-23), including the use of the "Reference Man" concept, the gastrointestinal tract model (absorption only from the small intestine, no endogenous secretion, exponential removal from each sequential compartment), and the ICRP 30 biokinetic models (distribution within compartments, growth of radioactive decay products) and the ICRP radioactive dose models (identification of source and target regions, spatial relationships between source and target regions). Specific uncertainties associated with radon gas inhalation include the uptake, retention and distribution of radon gas and the distribution of radon decay products in body tissues. The magnitude and direction of these additional uncertainties were not evaluated in USEPA (1989b). Overall, considering both model formulation and parameter variability, it was estimated that the uncertainty in dose was about a factor of 2 (USEPA 1989b).

Compared to external X- or gamma radiation, which gives a fairly uniform dose, internal emitters, particularly alpha emitters, give an irregular, rather uncertain dose distribution. The ICRP and RADRISK pharmacokinetic models assume uniform distribution of radionuclides in organ or tissues. This approximation is not necessarily true. While many studies have shown uneven distribution of radionuclides in tissues, there is not sufficient data on any radionuclide to establish a detailed pharmacokinetic model using non-uniform tissue or organ nuclide distribution. To the extent that the pharmacokinetic model is inaccurate regarding distribution within tissues, the doses calculated and the risks estimated will be inaccurate.

Bone exemplifies the dosimetry problems associated with alpha emitting isotopes. The short range of alpha particles in tissue and the high rate of deposition of energy make the distance from source to target cells a very important consideration. In bone, target cells for osteosarcoma induction are considered to be, and the dose is estimated for, cells up to 10 μm from bone surfaces. The selection of the thickness of the layer in the calculation of bone surface dose is somewhat uncertain. Harley and Pasternack (1976) calculated alpha dose rate for radium-226 and its decay products in uniformly labelled bone in the tissues outside the mineral bone. Noting the report of Lloyd (1970) of a 5 μm thick layer of osteoid between mineralized bone surfaces and cellular layers, Harley and Pasternack (1976) adopted a distance of 10 μm from mineral bone as appropriate for calculating endosteal dose. On the other hand, Scott (1967) indicated cell nuclei at a distance of 10-15 μm from mineralized bone surface in rats, and James and Taylor (1971) calculated dose out to a distance of 20 μm from bone surface. The International Commission on Radiological Protection (ICRP) recommended calculating endosteal dose rate over a distance 5-10 μm from the trabecular surface (ICRP 1968). Mays and Tueller (1964) calculated doses for Ra-226 out to 70 μm from bone surface and stated that the average dose rate for soft tissues from 0-10 μm was used because: (a) osteosarcoma induction appeared to occur in cells adjoining bone surfaces and (b) dose-rates were maximum at bone surfaces. They also reported the suggestion of Owen (1963) that cells located over a one-cell layer from bone surfaces were at risk. Vaugan (1972) had shown a 5 to 20 μm thick cell layer in rabbits and Lloyd and Henning (1983) demonstrated a single cell layer 0.6 to ≥8.1 μm thick in human bone. Thus, the assumption that the endosteal dose should be calculated for a 10 μm thick layer introduces some uncertainty into the assessment of risk from internally-deposited radionuclides and consequently into the prediction of risk from inhalation of radon gas.

Another aspect of bone dosimetry that is uncertain is prediction of leukemia due to irradiation of bone marrow. For leukemia induction, the possible non-uniform distribution of target cells in bone marrow is neglected in the models. Irradiation of bone marrow by low-LET radiation leads to leukemia in man and animals. Leukemia has also been reported in ThO_2 patients and is associated with enriched uranium exposure in animals (NAS 1988). However, radium dial painters with huge radium deposits in bone have not developed leukemias along with the radium-induced bone sarcomas, sinus and mastoid carcinomas. Nor have patients treated with Ra-224 developed an appreciable number of leukemias (NAS 1988). The evaluation of leukemia induction by high-LET radiation in the RADRISK model also introduces uncertainty into the evaluation of risk from inhaled radon gas.

The situation described for bone also applies, to a greater or lesser extent, to all tissues with a non-uniform distribution of radioisotope. This complicates direct application of a risk coefficients derived from X-ray or gamma exposure with a dose calculated using a pharmacokinetic model and increases the uncertainty in the assessment of risk from inhaled radon gas.

4. Genetic and Developmental Effects

No direct evidence is available concerning genetic or developmental effects of radon exposure in humans. Assumptions used to evaluate these risks are: that developmental effects cannot be calculated due to the lack of a plausible dosimetry for the developing fetus for internally-deposited radionuclides, that genetic effects of high-LET radiation can be calculated from genetic effects of low-LET radiation, that genetic effects of low-LET radiation are a stochastic (non-threshold) response to irradiation of gonads and that the radiation dose to gonadal tissue can be estimated as the dose to average tissue calculated in the model used to evaluate radon ingestion.

Lack of Quantification of Developmental Effects

Risk coefficients have been derived for developmental effects of external low-LET exposure (USEPA 1989b). The most substantial developmental risk is mental retardation from exposure during gestation weeks 8 to 15 (USEPA 1989b). For continuous exposure, assuming a uniform dose rate to all tissues, the risk per offspring corresponds to about 10% of the risk of genetic defects due to 30-year exposure of a parent. If the dose to the developing fetus from ingested and inhaled radon gas were similar to the dose to germ cells, then genetic plus developmental risks would be only about 10% higher than genetic risks alone. Developmental effects may exhibit a threshold for induction (USEPA 1989b), in which case there would be no increase in the calculated risks.

Basing Genetic Effects of High-LET Radiation on Effects of Low-LET Radiation

High-LET radiation has been shown to damage genetic material in somatic cells of exposed humans (USEPA 1989b). This provides evidence supporting the likelihood of genetic damage to germ cells from high-LET radiation, but does not provide a basis for independent assessment of this risk. The assumption that high-LET effects can be extrapolated from low-LET effects based on Relative Biological Effectiveness (RBE) is commonly made in radiological risk assessment, but the degree of uncertainty contributed by this assumption is difficult to quantify.

Non-Threshold Nature of Genetic Damage

The radiation protection community is generally in agreement that the probability of genetic change is linearly proportional to radiation dose and that no threshold exists for initiation of heritable damage to germ cells (USEPA 1989b). If such a threshold does in fact exist, and if this threshold exceeds the doses from environmental levels of radon exposure, then no genetic risks would be expected.

Estimation of Dose to Gonadal Tissue

The dose to gonadal tissue from ingestion of radon was assumed to be equal to the dose to general body tissue calculated by the model of Crawford-

Brown (1990). Partial support for this assumption is the close agreement between the dose predicted for gonadal tissue and general body tissue by the RADRISK model. To the extent that ovaries or testes may selectively take up or exclude radon, the calculated doses would be inaccurate. The previous discussions of dosimetric uncertainties in the Crawford-Brown (1990) and RADRISK models would also apply to calculations of general tissue and gonadal doses.

B. Parameter Variability

1. Cancer Risks from Radon Ingestion

The key parameters used in evaluating the cancer risk of radon ingestion are the amount of radon ingested as a function of drinking-water concentration, the values of organ-specific risk coefficients and the relative biological effectiveness of alpha irradiation.

Radon Ingested at a Given Water Concentration

Radon ingestion was evaluated assuming that radon would be lost from all beverages except direct tap water before ingestion. Nearly all radon is released from water heated to the boiling point (Partridge et al. 1979), and so coffee, tea, soups, etc. would not be expected to retain any radon. Such beverages account for the vast majority of tap water intake other than direct tap water (Pennington 1983, USEPA 1984, Ershow and Cantor 1989). Therefore, the assumption that only direct tap water contains radon probably is only a slight underestimate of radon intake for most individuals. Several analyses have indicated that the average consumption of direct tap water is about

0.7 L/day (Pennington 1983, USEPA 1984, Ershow and Cantor 1989). A value of 1 L/day was used in this assessment as a reasonable, protective value. The risks of radon ingestion are linearly proportional to drinking water ingestion rate, and the use of 1 L/day provides a standard value for which cancer risks are calculated for drinking water contaminants. The risk to a typical member of the population will be somewhat lower than those calculated, by about a factor of 1.4.

It was also assumed that 20% of the radon in direct tap water would be lost to the air in the process of filling a glass from the tap and drinking water from the glass. As discussed in Section IV.C, various values for the loss of radon from water have been reported: 13% to 14% (von Dobeln and Lindell 1964), up to 25% (Hursh et al. 1965) and 0% to 53% (Suomela and Kahlos 1972). An additional factor that would contribute to the reduction in systemic dose is the loss of radon through eruction due to degassing of cold tap water heated in the stomach (Gosink et al. 1990). Crawford-Brown (1990) used a value of 80% retention of radon in water (equivalent to 20% loss), but considered that the retention was not well characterized and assigned a GSD of 1.5 to this value.

Organ-Specific Risk Coefficients

Organ-specific risk coefficients used to evaluate the ingestion risk of radon were derived from values presented in the BEIR III report (NAS 1980b). The values that were used were mortality risks using the linear-linear model with a 10-year minimum latency and a 30-year follow-up using MCHS 1970 mortality data and a birth male:female ratio of 1.0511, constrained by total risk at all sites. This procedure was used to derive relative risk coefficients for fatal cancers except for leukemia and bone cancer, for which absolute risks with a 2-year minimum induction period and a 25-year expression period were calculated from the BEIR III absolute risk coefficients, with the leukemia and bone cancer risk coefficients combined using a relative biological effectiveness of 8 for alpha particles. Mortality to incidence ratios were derived from values provided by BEIR III. USEPA (1989b) estimated that uncertainty in these parameters from low-dose extrapolation was a factor of 2 or less, from the time and age dependent factors was a factor of 2, from extrapolation to the U.S. population was less than 10% and from dosimetry and sampling errors was roughly 10%. Crawford-Brown (1990) estimated that the GSD of the absolute risk coefficient was 6, due primarily to uncertainty in extrapolation to low doses and low dose rates.

Relative Biological Effectiveness of Alpha Irradiation

The RADRISK model uses a value of 8 for the relative biological effectiveness of alpha irradiation in all tissues. This value was derived from the value of 20 recommended by ICRP (1980) when comparing high-LET radiations to low-LET radiations at low doses and dose rates. The low-LET effectiveness at low doses and dose rates was reduced by a factor of 2.5 in the ICRP formulation. Since the USEPA linear risk model for low-LET radiation does not use a dose rate reduction factor, the appropriate relative biological effectiveness is equal to 20/2.5 = 8 (USEPA 1989b). Calculated risks from radon ingestion vary approximately linearly with changes in the relative biological effectiveness, and risks calculated with ICRP risk coefficients with a relative biological effectiveness of 20 are 1.6E-7 per pCi/L (Crawford-Brown 1990), very close to the value of 1.5E-7 per pCi/L calculated with USEPA risk coefficients and a relative biological effectiveness of 8 (see

Section VIII.B.2). Crawford-Brown (1990) assigned a GSD of 1.5 to quantify uncertainty in the numerical value of relative biological effectiveness. In a recent publication, Crawford-Brown (1991) derived an ingestion risk of 4E-7 per pCi/L using organ-specific doses predicted by Crawford-Brown (1990), low-LET organ-specific risk coefficients from the RADRISK model (USEPA 1989b) and a relative biological effectiveness of 20 (ICRP 1980). This 2.5-fold higher risk is the result of using the ICRP relative biological effectiveness with USEPA derived low-LET risk coefficients.

2. Cancer Risks from Inhalation of Radon and Radon Decay Products

The key parameters used in evaluating the cancer risk of inhalation of radon and radon decay products are radon air concentration as a function of drinking-water concentration, the conversion of air concentration in pCi/L to yearly exposure, toxicokinetic parameters for inhaled radon gas and subsequent decay products, values for organ-specific risk coefficients, the relative biological effectiveness of alpha irradiation and the risk coefficient for lung cancer risk as a function of exposure to radon decay products in air. Uncertainties in organ-specific risk coefficients and relative biological effectiveness were discussed in the context of estimated cancer risks from ingestion of radon. The other parameters are discussed below.

Transfer of Radon from Water to Air

As discussed in Section IX.A.3, inhalation risks due to radon in water supplies are evaluated assuming that a transfer factor can be used to relate air radon concentration to water radon concentration. The value used for this parameter is 10,000:1. Two independent analyses of the typical value and range of values for the transfer factor have been performed. Becker and Lachajczyk (1984) found that for typical ranges of ventilation rates, water-to-air transfer efficiencies, water uses and home volumes, the transfer factor ranged from 2,900:1 to 59,000:1, with a typical value of 14,000:1. Nazaroff et al. (1987) found that the transfer factor follows a log-normal distribution, and computed a geometric mean value of 15,000:1 with a geometric standard deviation of 2.88 (i.e., 95% of houses would have transfer factors in the range of 1,900:1 to 120,000:1). The arithmetic mean transfer factor was estimated to be 9,100:1 (Nazaroff et al. 1987). Uncertainty in the estimate of the mean or typical transfer factor is contributed by the representativeness of the available data, which cannot be quantified, and by the number of samples upon which the estimate is based, which contributes a geometric standard error of 1.06 (Nazaroff et al. 1987). Additional uncertainty is contributed by lack of independence among parameters (for example, the mutual dependence of house size and ventilation rate on the age of the house and the potential effect on all parameters of type of dwelling and number and age of persons in the household). Overall, the greatest uncertainty may be contributed by the lack of recent and representative data upon which to base estimates of national average values.

The transfer factor analysis computes an average concentration of radon in indoor air, and does not account for higher exposures during episodes of radon release from activities such as showering or laundry. An analysis by USEPA (1991b) indicates that the increased exposure to elevated radon air concentrations resulting from such activities can be up to a factor of 3, but typically is about 1.5. Certain individuals would be exposed to considerably higher radon concentrations (e.g., those who take frequent, long showers in a small, air-tight shower stall) and others would be exposed to considerably lower concentrations (e.g., those who take baths rather than showers, those who do no laundry) (USEPA 1991b).

The analysis of increased radon air concentrations in showers does not take into account the differences in the dosimetry of radon and radon decay products under showering conditions. The dose to the lung would be decreased because of the time lag involved in the build-up of radon decay products in air after radon gas is released from water. However, the dosimetry would also be influenced by the characteristics of the aerosols to which the decay products become attached. Current lung models assume that decay products are attached to solid particles that may become hydrated in the respiratory tract, and that are removed by mucociliary clearance (NAS 1991). It is likely that aerosol water particles produced during showering would have different properties in the lung, but no data were located to evaluate the extent or even the direction of the effects on lung dose at a given radon activity. This uncertainty about radon dosimetry during showering makes a relatively large contribution to the overall uncertainty concerning the inhalation risk of radon released from water.

Radon Exposure as a Function of Air Concentration

To calculate risk, radon air concentrations in pCi/L must be converted into exposure units. For inhalation of radon decay products, this requires values for equilibrium factor and hours per year spent indoors. For inhalation of radon gas, this requires values for breathing rate and hours per year spent indoors. The equilibrium factor used to convert radon gas concentration to exposure to radon decay products is 0.5 (USEPA 1989b). Although variations in the equilibrium factor have little effect on lung dose for a given total radon progeny concentration, the equilibrium factor does directly affect the conversion of radon gas concentration into radon progeny concentration (NAS 1988, 1991). The equilibrium factor can vary from house to house and within a given house due to air exchange, air movement, room surface area, aerosol particle size and other factors (USEPA 1991d). Measured equilibrium factors range from 0.02 to 0.83 (Toohey et al. 1987). USEPA (1991d) reviewed several recent studies of the equilibrium factor and found mean values ranging from 0.3 to 0.6. These data appear to support the use of 0.5 as a nominal point estimate of the mean equilibrium factor in American homes, with an uncertainty range of 0.35 to 0.55 (USEPA 1991d). An additional uncertainty is introduced in the evaluation of exposure to radon decay products due to radon released from water because an appreciable fraction of total exposure occurs during peaks of air concentration from radon released by showering, laundry, etc. (USEPA 1991b). The equilibrium factor in a shower is likely to be lower than in general household air (McGregor and Gourgon 1980), but sufficient data are not available to make a quantitative correction to the equilibrium factor used to calculate the typical exposure to radon decay products resulting from radon released from water.

An occupancy fraction of 75% is assumed (i.e., 18 hrs/day spent inside the home) for evaluation of risk from inhaled radon decay products and inhaled radon gas. The estimated average time spent indoors at home by adults is 16 to 17 hours per day (USEPA 1989a). Thus, 18 hrs/day may be a slight overestimate of the national average. Values for breathing rate vary from about 1 L/min for a sleeping infant to about 40 L/min for an adult performing heavy labor (NAS 1991). The average value of 22,000 L/day (15.3 L/min) used to assess exposure to radon gas appears to be a reasonable estimate of the average adult breathing rate in homes.

Uncertainty in the equilibrium factor contributes more than uncertainty in breathing rate or occupancy to the overall uncertainty in the conversion of air radon concentration to inhalation exposure to radon decay products and radon gas.

Risk Coefficient for Lung Cancers per WLM

The assumptions used to derive the risk coefficient for inhalation of radon decay products are described in Section IX.A.3. The central estimate of 360 fatal lung cancers per million WLM was derived by averaging the estimates from the BEIR IV and ICRP 50 models. The BEIR IV estimate is 305 per million WLM, with no numerical estimate of uncertainty, and the ICRP 50 estimate is 420 per million WLM with a range of 140 to 720 per million WLM. The ICRP 50 range was suggested to represent the range in uncertainty of the USEPA risk estimate due to uncertainty in the miner cohorts (USEPA 1989b).

The central estimate of risk was derived assuming that exposures in mines and homes resulted in equal lung doses and that a background radon exposure of 0.25 WLM/year contributes to national lung cancer mortality (USEPA 1989b). The adjustment for differences in doses in mines and homes has recently been reexamined by the National Academy of Sciences (NAS 1991), and USEPA may change the current ratio of 1.0 when this report is fully analyzed. The numerical value of the background radon exposure level may also change when results of the National Radon Survey become available. These potential adjustments are each likely to affect the risk coefficient by no more than 30%.

Toxicokinetic Parameters for Inhaled Radon and Decay Products in the RADRISK Model

Risks from inhalation of radon gas were evaluated using the RADRISK model. Toxicokinetic parameters include age-dependent metabolic parameters and organ retention of radon and radon decay products. Age-dependent metabolic parameters include organ size, organ uptake fractions and effective organ half lives. USEPA (1989b) did not specifically address the uncertainty contributed by age-dependent factors for radon in the RADRISK model. However, since risks are calculated for lifetime exposure, overall uncertainty will be less than the uncertainty in childhood risks, due to the relatively short duration of childhood. The RADRISK model uses models for retention of radon decay products derived from ICRP models for alkaline earth metabolism (Sullivan et al. 1981). USEPA (1989b) estimated that uncertainties in dose estimates were 2 or less for radon gas and lead and 5 or more for polonium. It is likely that the uncertainty for bismuth would be similar to that for polonium.

3. Genetic and Developmental Effects

The risks of genetic and developmental effects of radon were analyzed using the radiation dose for general body tissue calculated with the model of Crawford-Brown (1990) for ingested radon and the dose for gonadal tissue calculated with the RADRISK model for inhalation of radon gas. The same exposure parameters discussed in Sections IX.B.1 and IX.B.2 also influence the predicted genetic risk: the amount of radon ingested as a function of drinking-water concentration, radon air concentration as a function of drinking-water concentration, the conversion of air concentration in pCi/L to yearly exposure, toxicokinetic parameters for inhaled radon gas and subsequent decay products, values for organ-specific risk coefficients (in this case, mutation risk in gonads) and the relative biological effectiveness of alpha irradiation in causing genetic damage. Only the latter two parameters are specific to genetic effects.

Mutation Risk Factors

The Agency has derived risk factors for genetic effects of low-LET irradiations, based on estimates by expert committees evaluating data primarily derived from animals exposed to low-LET radiation (USEPA 1989b). The Agency identified six sources of uncertainty in the estimates for low-LET radiation risk factors: species used for extrapolation, genetic loci used for extrapolation, dose-rate reduction factor, relative sensitivity of oogonia and spermatogonia, spontaneous mutation rate and doubling dose in man. The true risk was estimated to be within a factor of 4 of the predicted risk for estimates of mutations in the first generation, with additional uncertainty introduced by extrapolation to the equilibrium (all-generation) rate of genetic effects (USEPA 1989b).

Relative Biological Effectiveness of Alpha Irradiation

The Agency based the RBE of alpha radiation in causing genetic effects on the conclusions of the BEIR IV committee that the RBE was 15 for chromosomal aberrations and 2.5 for all other effects (NAS 1988). The overall average RBE is then 2.7, which was rounded to 3 (USEPA 1989b). The uncertainty in the selection of RBE was estimated to be a factor of 5 (USEPA 1989b).

C. Summary

One of the largest sources of uncertainty in assessing the risks of radon in drinking water is the assessment of exposure to radon decay products in air resulting from the presence of radon in water supplies. Uncertainty in this assessment includes estimation of the transfer of radon gas from water to air, structural and human activity parameters influencing exposure in general household air and to peaks of concentration in showers and dosimetric assumptions used to extrapolate observed risks in underground miners to household exposure. There does not appear to be any systematic bias towards over- or under-prediction of risk, due to these or other assumptions and parameters. The uncertainty contributed by modelling assumptions and parameter variability in prediction of average population exposure may be a factor of 3 to 5 and the uncertainty in extrapolation of lung cancer risk may be a factor of 3. These considerations suggest that the true average individual risk is likely to be within an order of magnitude in either direction of those calculated for members of the general population.

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